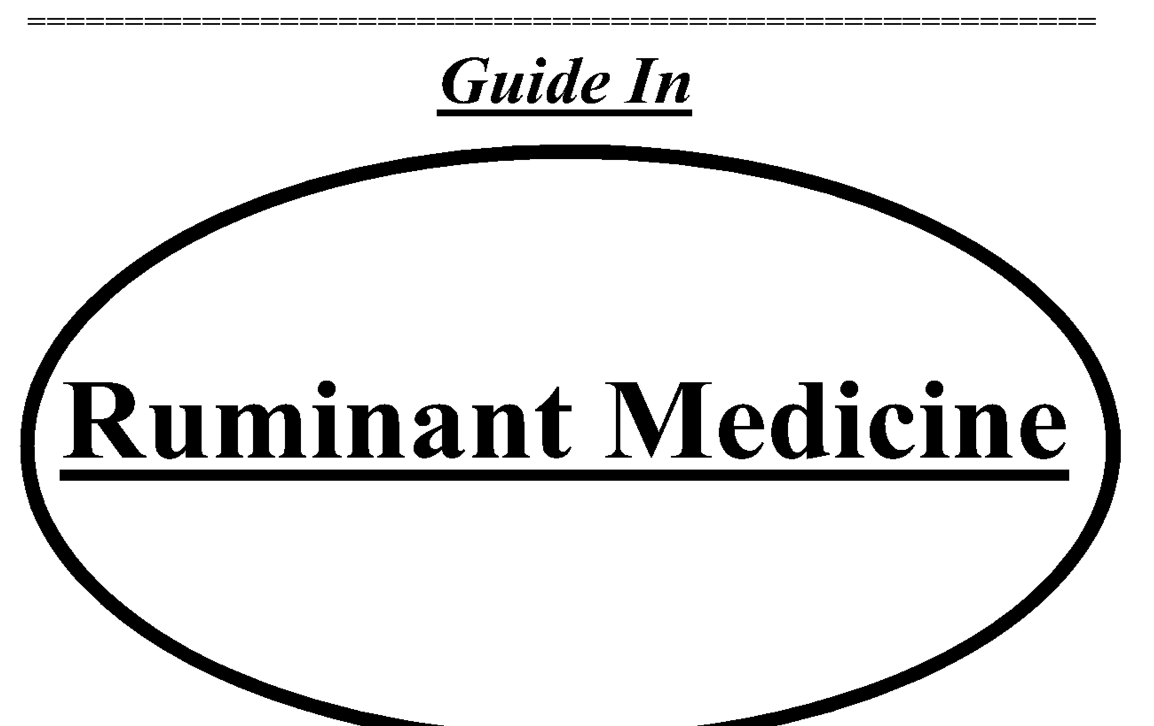
Guide In



Ruminant Medicine

(Sheep, Goats, Cattle, Buffalos and camel)

By

Abd Elghany Hefnawy

Introduction

Ruminant medicine is one of the most important branch in the veterinary medicine as the ruminants animals play an important role in the animal production and in the economic state of the country .so diseases that affecting the ruminants have great effect on the animal production as well as in the economic state of the country .

In this book (Guide In Ruminants Medicine) we try to highlighting on the most important medicinal problems which can occur in the ruminant and affect on the general health and productivities of the ruminants with explanation of the main etiological and predisposing factors of the diseases as well as pathogenesis, clinical forms ,methods of diagnosis and the lines of treatment and control of the most important medicinal diseases that affecting sheep, goat, cattle, buffalos, and camel.

This book consists main four parts, part (I)Small Ruminant Medicine (sheep and goat), part(II) Large Ruminant Medicine (cattle and buffalo) , part (III) Camel Medicine and part (IV) Some of general systemic,toxocological and allergic diseases.

We try in this book to be suitable for the students of the facilities of veterinary medicine, veterinarians as well as the students of the postgraduate studies.

Dr. /Abd El-Ghany Hefnawy

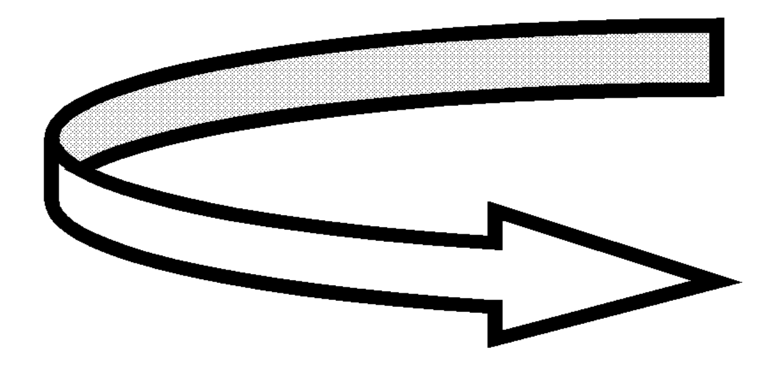
Dedication

I would like to thank all the members of my family, my father, my mother, my brothers and sisters as well as my wife Dr.Seham Youssef, my daughter Rewaa and my son Karim for their Permanente support and patient until completing this work .Also I would like to thank Mr. Samuel Gomez and his wife Esther Selene and their daughter Cesia for their support to me until completing this work in Mexico.

Part (I)

Small Ruminant Medicine

(Sheep and goats)



Chapter No.1

Metabolic diseases

1-Pregnancy toxemia

2-Hypocalcemia

3-Haypomagnesemia

1-Ovine Pregnancy Toxemia "OPT":

Synonyms:

1-Ketosis 2-Lambing sickness 3- Twin lamb disease

4-OPT "ovine pregnancy toxemia 5- Post parturient dyspepsia.

Definition:

It's one of most important metabolic disease of ewe due to disturbance in the carbohydrate and fatty acids metabolism which leading to hypoglycemia, ketonuria and ketoneamia and decrease in the level of the liver glycogen.

Occurrence:

1-Ovine pregnancy toxemia (OPT) may occur in all breeds of sheep and goats in the second and sub-sequence pregnancies more common in aged or senil animals (5-10years old).

2-Thin or over conditioned ewes bearing twins or triples develop the disease during the last months of pregnancy i.e. 4^{th} months.

3-The disease is probably occurs in all sheep producing countries but the prevalence is high in New Zealand, USA. Britain and South Africa.

4-The disease usually associated with nutritional disturbance as low carbohydrates in diet specially in the late stage of pregnancy.

Etiology and pathogenesis:

1-Rapid growth of twine or triples fetuses

2-Declining or interruption of the pregnant ewe nutrition which leads to OPT.

3-Psychological stress as transportation or fear.

4-In the last 6 weeks of pregnancy the fetus need about 70-80gm glucose/day while the dame require about 85-100gm glucose/day ,so inadequate supplementation of glucose in late pregnancy leads to OPT. or Hypoglycemia.

Compensatory roles of ewe against hypoglycemia:

1)-Increase consumption of grains as exogenous source of glucose.

2)-Maternal tissue reduce it's consumption of glucose to provide the glucose requirement for fetuses.

3)-Maternal tissue hydrolyse the storage fat (i.e. increase the fat catabolisme) to give glycerol, which converted into glucose,(gluconeogenesis) and fatty acids oxidation leading to produce of large amount of ketone bodies (Actone, Acete-acetic acid & B-hydroxyputeric acid) leading to ketonuria and ketoneamia.

4)-The stress and low caloric intake of OPT have a profound effect on the kidney and adrenal medullary gland with a significant reduction of the renal blood flow and glemorular filtration rate which raises plasma renine activites and elevate plasma cortisol level. That manifested post mortamly by enlarged adrenal gland.

5)-The cortisol level may inhibit glucose utilization by the tissue and causes clinical manifestation (especially neurologic manifestations) which

associated with OPT.

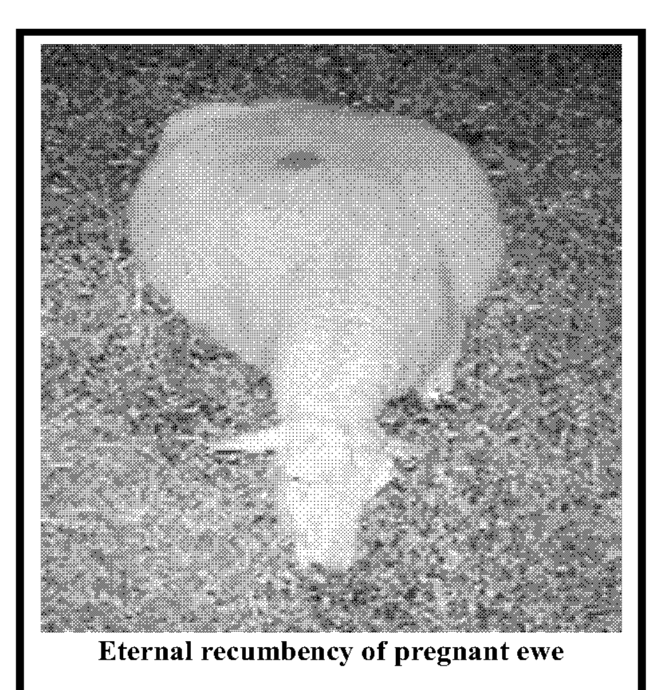
6)-Hypoglycaemia increase → fat catabolism increase → accummulation of lipid predemontally Tri-acyle glycerol which responsible for fatty liver (P.M.).

Clinical Signs:

1-The affected animal is thin or bese and course of the disease about 2-10 days.

2-Isolation from flock and declines feed (anorexia)

3-In coordination in movement, elevation of head and pricked ears that give what so called "*listening attitude*" or "*star gazing position*"



Eternal recumbency of pregnant ewe

4-Sternal recumbancy and rises only by assistance.

5-Accelerated respiration. with mucous nasal disarge.

6-Neurological signs such as blindness, muscular tremors, convulsions coma and finally death.

7-Morbidity rate about 20% of flock and mortality rate may reach 80% of the diseased ewes.

8-Urenic or acetan adour of the mouth.

Diagnosis:

-History of late stage of pregnancy. of 2nd. or subsequent pregnancy and old or senil animals.

I)-Case history with clinical signs. The late stage of pregnancy.

***II)-PM lesions*:**

1-Thin or over-Conditioned ewes. 2-Uterus containes 2-3 fetuses

3-Fatty liver(fraible liver and pale yellow in color)

4-Enlarged adrenal gland by as much as 65%.

5-Aceton adour of the carcase.

III)-Lab diagnosis:

1-Hypoglycemia the glucoge level is below 25mg% (Normal = 40 – 60 mg%)

2-Ketonaemia 10-1000mg% (Normal = up to 10 mg%)

3-Ketonuria 80-1300mg% (Normal = up to 50-70 mg%)

4-Milk ketons 40mg% (Normal = 3 – 10 mg%)

5-High level of plasma non-protein nitrogen in the termal stage due to decomposition of lambs (fetuses) – ↑level of BUN and creatinine.

6-High level of cortisone is diagnostic in ewe.

7-Iron, magnesium, potassium → low level.

8-*Rother's test*:

Used for detection of ketonuria as the following:

5 ml of urine of suspected case.

-Amonium sulphat until fully saturation of the urin sample

-1 ml of cancentrated ammonium solution on the wall of tube.

-Few drops of sodium nitro prosside. ----→Voilet ring appears in positive case

***Differential diagnosis*:**

***With Hypocalcemia. In whcih`.**

1-The animal take "*Frog attitude*" lateral recumbence and head is reflected on the shoulder toward the flanck region with extension of the hind limbs.

2-Response to calcium administration.

3-Low calcium level (N = 8-12mg%)

4-Negative rothers test.

Treatment and prevention:

1)-Correction of hypoglycaemia by injection of glucose preparation I.V. (R/Dextrose 5, 20,or 40% (250-500ml).

2)-Correction of dehydration and acid-base imbalance by injection of fluid therapy (R/-Ringers lactate or normal saline or sod. Bic. I.V.)

3)-Stimulation of depressed appetite by giving of stomactic (R/-Stamatone-Digestin-Uccamdigest-Veta digest………..etc.)

4)-Oral Propylene glycol or Corn syrup as source of energy (200ml 4 times daily along with 3-4 lites of fluid therapy.)

5)-Adminestration of vit B-complex or Calcium to improve rumen

motility "50-125ml 20% Ca^+ borogluconate"

6)-Feeding on concentrate or hand feeding may be necessary.

7)-Surgical interference by cesarean section.

8)-Glycerol 4 oz. (120ml glycerol + 120ml worm water erally) i.e. 50% glycerol.

Control:

1-Avoiding overfattening or starvation specially during late pregnancy.

2-Prohylactic dose of sodiume propionate as it actes as Anti-ketogenic and it's glucogenic substances.

3-Excercising of ewe specially in late pregnancy.

4-Avoiding stress factors as transportation………etc.

5-Feeding on concentrate contains 10% protein (0.25kg/day and increased 1kg/day at late 2 weeks give good protection.)

6-Ground maize has particular quality in providing readily available glucose in ration and is a logical preventive feed. Due to it contains good amount of alpha-polemerized glucose which notserminted in the numen and pass to the intestine and absorped there.

2-"Hypocalcaemia" "HC"

Synonyms: 1- Milk fever 2- Trasport tetany "parturent paresis"

Definition: It's acute metabolic disease of sheep characterized by tetany in coordination, paralysis and coma, caused by an inadequate supply of metabolizable calcium.

Occurrence: 1-Occurs in all breeds, sexes and ages of sheep beyond weaning age "Lactating and pregnant ewes, ram and feed lot lamb"

2-More common in last 6ws, of pregnancy or 1st week of lactation. In spite of after fasting or transportation of ram or feed lot lambs.

3-Geogrophically "H.C." occurs in all sheep producing contraries but Australia, New Zealand, USA, Britain and South Africa have high incidences.

4-Hypocalcemia occurs as a result of increase in :-

*The rate of calcium movement out of plasma with decrease in calcium absorption in the gut and decrease in calcium resportion from bone.

1-Calcium level is controlled by (parathyroid harmon; vit-D_3 and Thryocalcitenon and magnesium.)

2-Vit D_3 and parathyroid hormone is essential for:

A)-Absorption of calcium form intestin

B)-Resorbtion of calcium from bone.

C)-Reabsorption of calcium from renal tubules

3-Low level of magnesium leads to refractorness (resistance) of the bone to the action of parathyroid hormone as it acts for resorption of calcium from the bone so “HC” usually associated by hypomagneamia.

4-Females which have multiple fetuses are higher susceptable than that which have single fetuse that due to twince or triple fetuses need twice or triples times of calcium and phosphorus for mineralization of the fetuse skeletone than that needed for one fetuse.

5-Hydroxy proline is very important in skeletal metabolism and bone resportion but level of hydroxy proline is decreased with old age .so the old aged animals are highly susceptible than young.

“Etiology and pathogenesis”:

1-Normal calcium level is 8-12mg% while pathological level 3-6mg% and there are many factors predisposing of low level of Ca as:

A)-Prolonged consumption of calcium deficient diet which may lower the plasma calcium by 15-35%.

B)-Fasting from 2-6 days, especially when combined with transport which may lower the plasma calcium by 17-25%.

C)-Late pregnancy (last 6ws) or early lactation (1^{st}. 10day) may lower the plasma calcium by 50-60%.

D)-Presence of calcium depressing factors as administration of epinephrine or consumption of oxalate containing feeds… … … etc.

2- Presence of calcium depressing factors together with inability of the animal to mobalize the calcium rapidly from skeleton leading to serious

and fatal "HC".

3-Stress factor → increase of cortico steroid and epinephrine secretion which lead to :- *A)-Antagonizing of vitamin D_3 which is essential for calcium metabolism. B)-Decreasing renal resorption of calcium*

C)-Decrease intestinal reabsorption of calcium

D)-Inhibit the action of parathyroid hormone on the bone.

E)-Decrease the calcium absorption.

F)-Decrease the mobilization of calcium from bone.

All the mentioned processes may lead to increase calcium losses → HC.

4-If the calcium level reached dangerous level near 3 to 6mg%, irritability develops, followed by depression and coma and animal except for calcium treatment most animals die.

5- Ration high in "Calcium" and low in "phosphoros" in late pregnancy.→ depression the activity of parathyroid gland → "H.C"

6-Acute sever enteritis may leads to "H.C." due to impaired absorption from the intestine.

<u>Clinical signs:</u>1-In all classed of affected sheep abrupt clinical signs preceded by decrease in ruminal motility and uterine tone in case of pregnant ewe. 2- Cervico-vaginal prolapse with complete or non-dilated carvix.

3- In early stage there is stiffness and in coordination. And spradd led stance, especially in the hind limbs.

4-Later stages there are muscular tremors, muscular weakness, apprehension and rapid breathing. 5- Normal body temperature.

6- Ewes in advanced stage fall to lateral recumbancy and the hind limbs are extended back wardly "*Frog attitude*"

7- Death occurs within 4-48 hours if not treated by calcium.

8- Morbidity rate in feed lot lams 2-3% and the animals usually die unless they are treated. ***PM*** → There is no gross or microscopic lesions are discernible.

Diagnosis:**I)-Case history and clinical signs.**

II)-Laboratory examination

1-Calcium level lower than 8mg% (N = 8-12mg%) 2

2-Phosphorus may lowered than 4mg% (N = 7-9mg%) 1

3-Magnesium may lowered than 2mg% (N = 2-5mg%) ½

4-Eoesinophilia, neutrophillia and lymphopenia due to the adrenal cortically hyperactivity.

III)-Therapeutic diagnosis **i.e.** response to calcium therapy as treatment.

VI)-Differential diagnosis:-

1-Hypomagneseamia. 2-Enterotoxemia 3-Ketosis or "OPT"

Treatment and prevention:

1- Most sheep affected by "HC" give good response to calcium treatment 50-100cm calcium gluconate I.V. or s/c.

2- Avoiding all mentioned etiological and predisposing factors.

3- Massaging of hind limbs and avoid milking at least for 48 hr.

3- Hypomagnesemia "H.M."

Synonyms:

1-Grass tetany 2-Grass staggers 3-Wheat pasture Gpoisoning.

Definition: It's an acute metabolic disease of pastured sheep and goats characterized by excitative bearing (Hyperathesia) and spasdic tetany (clonic convulsion) caused by a depressed supply of metabolizable magnesium from prolonged consumption of lush immature grasses.

Occurrence: 1-H.M. occurs in all breeds of sheep and goats specially lactating ewes that grazing in early spring pastures.

2- More common in the first 4 weeks of lactation.

3- All classes of sheep and goats are susceptable i.e. ram, feed late lambs ,kids …….etc.

4- H.M. usually occur during spring and autumn following rains and rapid growth of grasses, including young wheat plants and other crops (usually within 1-3 ws after beginning of grazing).

5- Geographically the disease probably occurs in all sheep producing countries where grass and wheat pasture is an important part of sheep economy (USA. Australia, Britain, Netherlands, Scandinavia, Canda) the majority of cases occurs in the wheat bett of the high planis.

Etiology and pathogenesis:-1-The normal plasma level of magnesium and calcium is 2-3 mg%, and 8-12m% respectively.

2-The calcium and magnesium homeostasis is controlled by a complex

mechanisms under the influence of parathyroid hormone and vit. D3.

3-Various factors that may contribute to a decrease in serum magnesium. Concentration as:

(A)-Nor epinephrine and corticosteroid hormones.

(B)-Increased mineral corticoids concentrations.

(C)-Increased glucagon, and decreased carbohydrate intake.

(D)-Increased the dietary potassium intake and excessive water intake.

(E)-Exposure to cold a long with in adequate energy intake synergistically reduce plasma magnesium level (cold stress alone has a little effect on plasma magnesium level).

4-The plasma magnesium level normally increased in late stage of pregnancy and apruptly decrease during lambing and continue in decline for 2-3ws and ewes with twins have lower magnesium level for 2-3weeks, after lambing than ewes with single.

Pathogenesis: (1)-The pathogenesis of HM begins with lush grasses, including young wheat plants which rapidly grow after spring and autum rains. (2)-These plants especially which growing on ammonium-fertilized soil contain high concentration of protein, potassium and Amonia

(3)-Absorption of ammonia by the plants reduce uptake of calcium and magnesium with little effect on potassium, and produce high amide.

(4)-All mentioned factors (No.1,2 and 3) combined in the animal to create:

(A)-High concentration of ammonia in the rumen.

(B)-High level of rumenal PH. *(c) -Depletion of carbohydrate.*

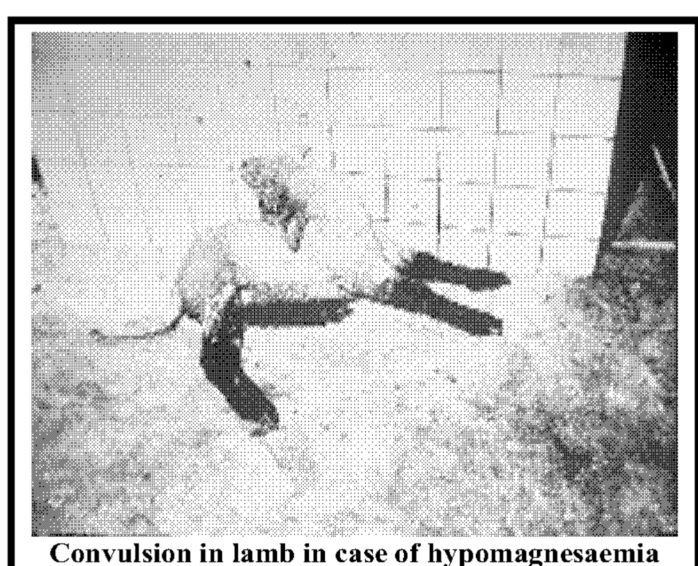
(D)-Reduction of availability of calcium and magnesium.

(5)-The adult ewe has a stage of magnesium in the bone which can be mobilized into extera cellular fluid and these mobilizations commence when serum magnesium level falls below 1.8mg% and when this storageis exhusted the magnesium level falls.

(6)-When magnesium level declined below 0.7mg% irritability develops and when level is 0.5% fatal tetany and convulsions ensue (resulted).

(7)-Death usually due to respiratory failure and the recovered animals are susceptible to recurrence of the disease.

Clinical Signs:



Convulsion in lamb in case of hypomagnesaemia

1-Clinical signs are of central origin and related to reduction in magnesium of the cerebrospinal fluid.

2-Hyperathesia (hypersensitivity to external stimuli as noise or light….etc).

3-Accelerated breathing – Tremors and in coordinated movement.

4-In advanced cases there are convulsion, recumbancy and coma then finally death.

5-Before the muscular convulsions, the body temperature is normal but

during convulsions the temperature may up to 43°c due to muscular contraction (clonic convulsions).

6-For mammals in general, the following proportionally expression applies:

$$\text{Irritability } \alpha \frac{[K^+]+[Na^+]}{[Ca]^{++}+[Mg]^{++}+[H]^+}$$

i.e. Irritability in sheep is directly proportional to the sum of the concentration of potassium and sodium while inversely proportional to the sum of calcium, magnesium and hydrogen ions concentration.

7-Morbidity rate about 20%.

8-Mortality rate up to 80% of affected untreated cases.

9-Course about 2-24 hours.

10-PM :- Petichial and ecchymotic hemorrhages commonlly on the serosal surface of the heart and intestine but the specific lesions are absent.

Diagnosis:

1-History and clinical symptoms.

2-Laboratory diagnosis (magnesium level about 0.50-0.25mg/dl)

3-Differential diagnosis with:-

(A)- *Hypocalcemia in which* (1) Calcium level below 4mg%.

(2) Animal gives response to calcium administration

(B)-*Enterotoxamia in which* (1)-Sudden deat (2)-High body temperature.

(3)-Presence of toxins in the intestine.

Treatment and prevention:

1)-Successful treatment of HM mainly depend upon early diagnosis and prompt intravenous injection of one of indictable products of HM which contains: Calcium brogluconate, Dextrose and magnesium boroglucanate as R1: Cal. D. Mag & Cal bor mag – Cal phormag….etc.

2)-Vit D3 which helps in absorption and mobalizsation magnesium as R/ D-Varel injection

3)-Supportive treatment as fluid therapy and dextrose. Muscular relaxatn for hyperthesia as: R/Neurazin- or chlropromazine……..etc.

Prevention: Magnesium supplement especially in spring and autumn seasons, and the supplmental magnsium may be provided through animal mixture containing.

-400 kg Calcium carbonate. -200kg trace mineral salt.

-150kg Magnesium oxide -200kg ground corn or other concentrates.

-50 kg dehydrated molases.

* 65kg lactating ewe requires from 43 to 96mg/magnesium daily. i.e. 7-14gm of the above mineral mixture (i.e. 2.5gm/10kg bwt).

Chapter No.2

Nutritional Deficiency Disease

1-Iodine deficiency”
2-Cobalt Deficiency “CD”
3-Iron deficiency
4-Vitamin-D Deficiency
5-Vitamin E and/or Selenium deficiency
6-Zinc Deficiency
7-Copper Deficiency
8-Hypovitaminosis-A

1- Iodine Deficiency "Goiter"

Definition: It's sub-acute or chronic deficiency disease characterized by enlargement of thyroid gland ,caused by deficiency of Iodine.

Economic losses due to:

1-Unthriftiness and low reproductive efficiency.

2-Fetal and lamb death and poor quality of wool.

Occurrence: 1-Goiter develops in all breeds, sexes, and ages but more frequent in ewes than rams and more frequently in fetuses and lambs than n yearling and adult animals.

2-Geographically more common in Iodine deficient soil asin USA, Canda Latin America, Venezula and Columbia & New valley in Egypt.

Etiology:- 1)-Inadequate intake of Iodine in Feed and water.

2)-Excessive consumption of goiterogenic compounds in plants as in cabbage, syabeans and peanut or turinp.

3)-Excessive calcium intake hinder the absorption of Iodine

Pathogenesis: 1-Ionized iodine is absorped from gastric and jejunal mucosae into the plasma then distrebuted to all tissues and actively concentrated and storaged in the thyroid gland.

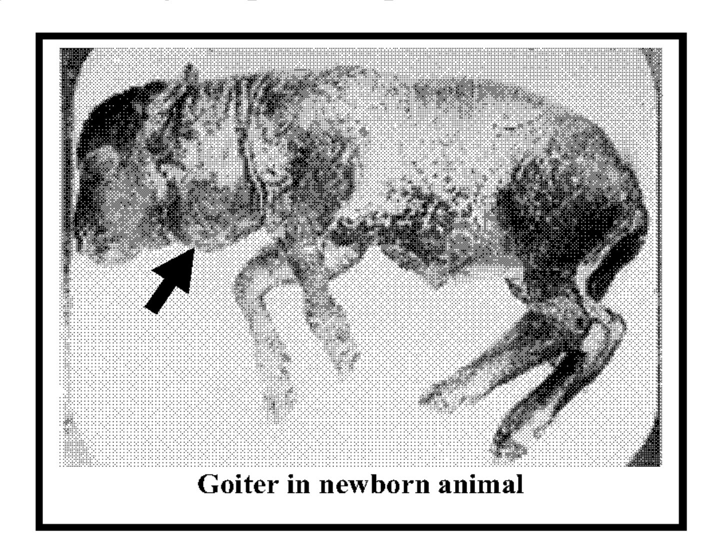
2-Within gland follicles ⟶ 'Iodine + tyrosine (Monoiodotyrosine-diiodotyrosine.)

3-Mono- + diiodotyrosin ⟶ triodotyrosine + thyroxine.

4-Thyroxine + globuline → throyoglobuline "that accummulatedin the colloid"

5-Due to Iodine deficiency in the diet the thyroid goland tend to compensate this by increasing the activity with the resulting in hypertrophy and enlargement of thyroid gland i.e. goiter.

Clinical Signs:



Goiter in newborn animal

1-Low growth rate due to low basal metabolic rate (BMR).

2-The neck is variably enlarged in the laryngeal area. (area of the throat)

3-Wrinkled skin and poor quality wool (sheep) or hair (goat)

4-Adult rams show reduced libido and low semen quality.

5-In ewes it show → Abnormal conception rate & irregular estrus and →Abortion or produce goitrous still born and → weak lambs.

6-Affected lambs show → thick, scaly skin with sparse wool, and → the neck is thickened, in some cases bulging.

7-Dyspnoea and noisy breathing with dysphagia and dysphonia. Due to pressure or the area of throat.

8-Localized or generalized alopecia

PM lesiones: 1-In adult sheep (Bilateral enlargement of gland, odema in and around the gland)

2-In neonatal lamb → enlarged and odematous thyroid gland.

3-Body cavities may contain excessive fluids.

Histologically: *Follicles lined with tall hyper plastic cells that invaginated into the lumen and colloids may be absent.

Diagnosis:1-History, clinical signs and PM lesions.

2-Differential diagnosis (vit A deficiency, Neoplasm or other causes of abortion).

Prevention and treatment:1-Providing adequate amount of dietary goiter.

2-Salt mixture with level of 0.5% of the diet and have approximately the following composition :-

Nacal	97%	Cabalt	0.015%
Copper	0.023%	iodine	0.070%
Iron	0.117%	manganese	0.225%
Sulfur	0.040%	zinc	0.008%

3-Addition of potassium Iodide.

4-Lugol's solution /drop/5kg bwt. And the high dosage is contra indicated that may leads to necrosis of follicular cells.

2-Cobalt Deficiency "CD"

Synonyms:

(1) Pining (2) Bush-sickness (3)White liver disease (WLD)
(4)Coat disease (5)Enzootic marasmus (6)Salt sickness

Definition:

It's chronic wasting disease of sheep due to cobalt deficiency which leading to defecate in synthesis of vit. B12 characterized by anorexia, anemia, and loss body weight.

Occurrence:

1-Young sheep and goat are highly susceptible than adults.
2- Weaned lambs and kids are highly susceptible than adults.
3- Cobalt deficiency affects ruminants grazing forage low in cobalt content.
4- White liver disease "WLD" occurs in area of sandy or pumice soils that deficient in cobalt.

Etiology and Pathogenesis:

1-Mainly due to deficiency of cobalt in diet result in deficiency of vit B12.
2-The only known function of cobalt in sheep nutrition is to promote. Vitamin B12 synthesis by the rumen micrfolara, so deficiency in dietary cobalt leads to inadequate production of vit B12 and ultimately a failure in enzyme systems.
3-Presence of sufficient amount of cobalt in the rumen leading to

synthesis of about 600-1000ug vit B12/day, so inadequate amount of cobalt leads to vit B12 deficiency.

4-The essential defect in cobalt deficiency in ruminants is an inability to metabolize propionic acid, which is accompanied by a failure in appetite and death from inanition (malnutrition).

5-The pathogenesis of ovine white liver disease is uncleared until now and, it has been proposed that a plant toxin and mycotoxine may be involved.

Clinical Signs:

1-The signs appeared within few months after feeding on cobalt deficient diet for long period.

2-In early stage (Anorexia, and anemia (mainly In appearance)- Loss of body weight.

3-The sheep become weak and emaciated.

4-Pale coloured mucous membrane and skin.

5-Profuse lacremation which stains the face.

6-Some animals may develop sever diarrhoea and if the animal not treated it may die.

7-Sub-clinical manifestations are

(A)-Lack of thrift.

(B)-Infertility in male and abnormal oestrus in female .

(C)-Decrease milk and wool production.

(D)-Neonatal mortality.

(E)-Reduce the resistance against disease.

8-In white liver disease all mentioned signs are involved in about 10% of the cases with photosensitization.

9-In seriously deficient region:

(A)-Morbidity rat 60%. *(B)-Mortality rate 80%*

(C)-Course of the disease is several weeks. to few months and the signs appeared after sheep grazing 4 to 6 months on cobalt deficient regions.

10- PM. Lesions :

(A)-Emaciated carcases *(B)-Atrophy of bone marrow.*

*(C)-Fatty degeneration of the liver *Haemosidrosis of spleen.*

Diagnosis:

I)-History, clinical signs and postmortem lesions.

II)-Laboratory diagnosis.

*(1)-Low level of haemoglobine 8-9gm/dl (N=11-12gm/dl).

* (2)-Decrease the amount of vit-B12 in the feces because the vit. B12 level in the feces is an indicator of the cobalt status of sheep and the following equation used to detected the amount of cobalt in take.

$$Y = 0.0779 X - 0.0757.$$

Y = the amount of cobalt in take. Expressed by mg/kg of dry feed

X= the amount of vit. B12 in the feces expressed by mg/gm of dry feces.

* (3)-Generally after trails each gm of feces contain 2,13 mg of vit B12 is indication of 0.09 mg of cobalt/1kg of dry diet. So detection or

measurement of vitamin B12 in the feces can be used to monitor the adequate amount of cobalt in take.

* (4)-Detection of plasma cobalt level (N = 0.17 – 0.5/µmol/l) = (N = 1-3 µg/dl) while in deficient animals these are reduced to 0.03 – 0.41 µmol/L.

* (5)-Vit B12 plasma level in cobalt deficient animal is below 0.20 mg/ml.

* (6)-Hypoglycemia (below 60mg% of glucose) and low level of alkaline phosphatase (less than 20 Iu/liter) with high level of ketone bodies.

Prevention and treatment:

1)-Supplementation of cobalt in:

-On water with range of: 0.2 – 0.4 mg cobalt/liter of water.

-On feed with range of: 2mg/cobalt/1kg of dry feed.

2)-Using of cobalt sulfate or cobalt chloride in salt mixture with average.

2.5gm cobalt/100kg of salt mixture (providing of 10gm salt/day will provide 0.25mg cobalt).

3)-The use of commercially available cobalt pellets will provide adequate cobalt levels up to 3 years provided they stay in the rumen and not regurgitated.

4)-Application of 5-3kg hydrated cobalt sulfate/hecta in the plants. to deficient soil will increase the cobalt level for 3-5 years.

5)-Daily dietary supplement of 1mg of cobalt/animal until improvement occurs.

3-Iron deficiency

Etiology:

1-Mainly primary due to deficient diet of iron. Specially in young animals which depend mainly on milk feeding (milk is deficient in Iron). 2-Continued blood loss by haemorrhage leads to anaemia and iron deficiency. 3-Heavy infestation of external or blood suckling parasites

Pathogenesis:

1-More than 50% of body iron present as constituent of haemoglobine and relatively small amount present in myoglobulin and certain enzyme which play a role in the oxygen utilization.

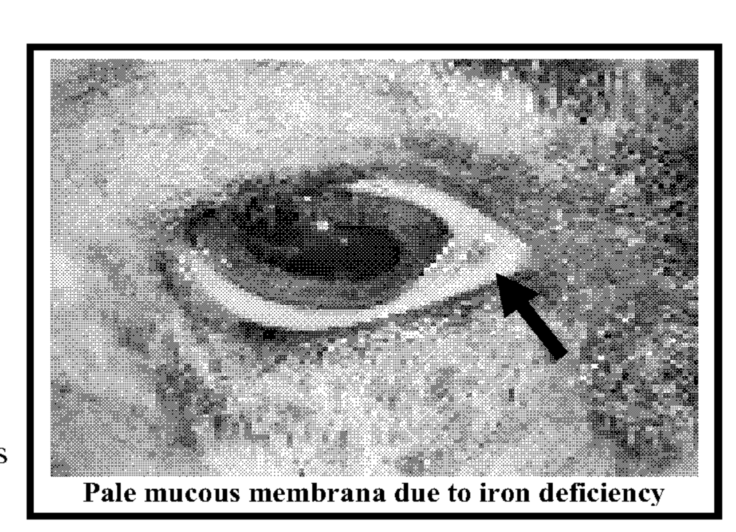
2-Daily requirement of iron about 30-40mg while animal that fed on milk only the iron milk about 5mg/day, so it leading to iron deficient anaemia specially if the animal not supplmented by iron in diet.

Clinical Signs:

1-Usually appear in 3 weeks of age [suckling lambs or kids]

2-Low growth rate and anaemic animals.

3-Food intake is obviously reduced.



Pale mucous membrana due to iron deficiency

4-Diarrhoea more common but normal in colour.

5-Dyspnoea (due to anaemic hypoxia), increase the heart rate specially after exercise.

6-Pale coloured mucous membranes due to anaemia.

7-Alean, white hairy look is probably more common.

Diagnosis:

1-History and clinical signs.

2-Laboratory diagnosis to detect low level of haemoglobin (N =11-13gm%).

3-Preventive diagnosis by iron administration.

Treatment:

1-Administration of organic iron preparation as (Iron – dextran/ Iron-sucharate/ Iron-gluconate).

*Dose 0.5-1gm/day/weak.

2-Oral treatment by iron gluconate or iron sulfate for 2 weeks 2-4gm/day.

3-Vit B12 (Cyanocobalamine) also can be given 5000µg/week singl dose.

4-Vitamin -D Deficiency

Synenomes

(1)-"Osteo dystrophic diseases" (2)-"Rickets or osteomalicia"

Etiology:

1-Primary cause: → due to deficient vit-D in food in take.

2-Secondary cause: → due to other cause as increase caroten in take.

3-In sufficient solar irradiation of the animal (uttravoilet rayes)

4-Heavy coat animal. Acts as predispose due to it insulate the skin from solar irradiation. Or heavy infestation with GIT. Nematodes.

5-Toxicity of melybodnum, or copper chlorid → osteo destrophic disease.

Pathogenesis:

1-Vitamin-D is a complex of substance with anti-rachitogenic activity, and the important component are as following:

(A)-Vit-D3 (cholecalceferol) is produced from its precursor 7-dehydro-cholesterol in mammalian skin and by natural irradiation with ultreavoilet light.

(B)-Vit-D2: is present in sun-cured hay and is produced by ultra voilet irradiation of plant sterols.

**Calciferol or viosterol is produced commercially by the irradiation of yeast (Erogosterol is the pro-vitamin).*

(C)-Vit-D4-D5 →formed naturally in the oil of some fish.

2-Vitamin D which produced in skin or ingested in the diet and absorped in the intestine is transported to liver where it produced into 25-hydroxy cholecalciferol which transported to the kidney in which at least two additional derivatives are formed by 1-alpha-hydroxylase Enzyme.

a-1,25 dihydroxycholecaciferol (DH.CC)

b-24,25 dihydroxycholecaciferol (DH.CC)

3-In case of calcium need or deprivation the most predominant form that produced in kidney is 1,25- DH.CC (metabolic form of vit-D) which very important in:

(A)-Transportation and absorption of intestinal calcium.

(B)-Regulation of phosphorous absorption and metabolism specially it's loss from the kidney.

(C)-Mineralization of bone.

So any deficiency of vit. D leading to disturbance in calcium and phesphrous absorption and metabolism.

4-When calcium phosphorous ratio is wider than the optimum (1:1to 2:1) so the requirement of vit-D for good calcium and phosphorous retention and bone mineralization is increased so, any deficiency of vitamin-D leading to the disease.

5-The minor function of vitamin D include maintenance of efficiency of food utilization and calorigenic action so the metabolic rate is depressed when the vitamin is deficient.

6-Some evidence suggests that vitamin-D may have a role in the immune

system so, it's deficiency leads to decrease in the animals resistence and become susceptible to be infected by many diseases.

Types of osteoperosis:

1-Matrix osteoperosis (bony atrophy)

2-Rickets

3-Osteomalicia or soft or mineral osteoperosis or bone softness.

4-Stunted bone growth.

1-Matrix osteoperosis [bony atrophy]:

Affect the adult and young animal and it characterized by:

----Low amount of protienetious substances of the bone per unite although good meniralization.

2-Rickets:

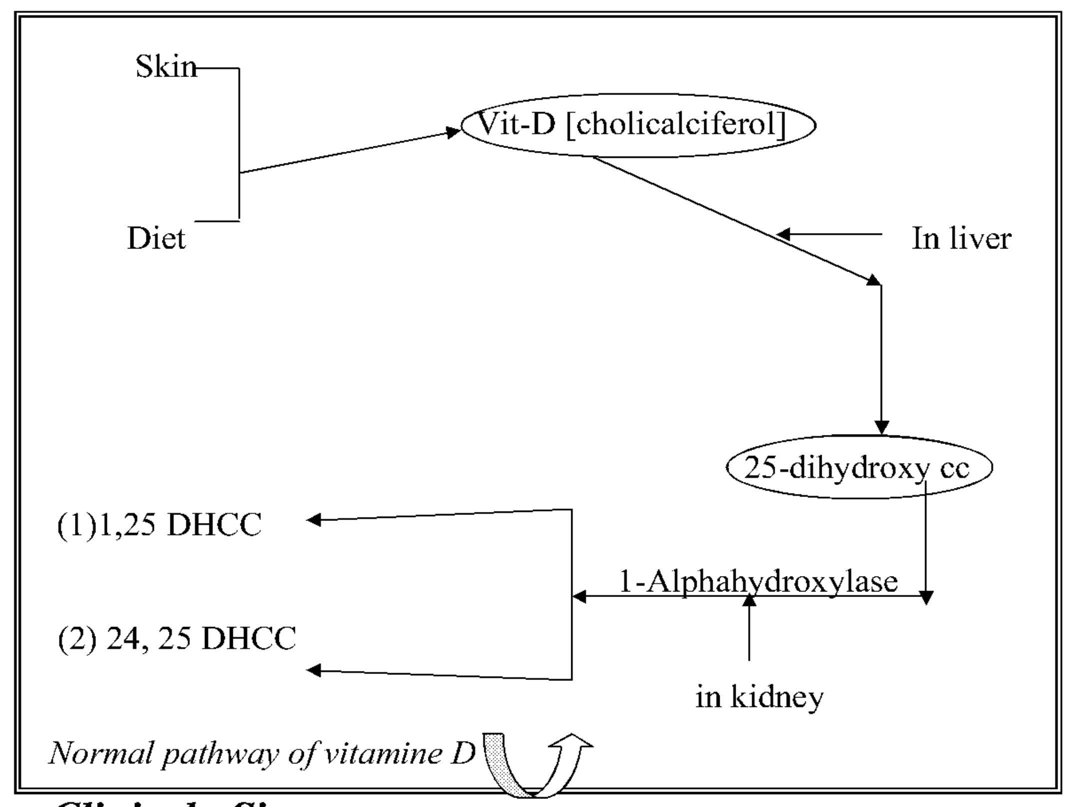
-Affect only young or growing animals this due to defect in the minealization although matrix is present normally.

3-Osteomalicia or soft, mineral osteopetrosis or bone softness:

This occurs in adult animal, the defect near in mineralization although there are normal proteineious matrix.

4-Stunted bone growth:

Due to defect in both protenious matrix and mineralization that lead to lower and abnormal bone growth usually in young animals.



Normal pathway of vitamine D

Clinical Signs: 1-Reduced the productivity of the animal & reproductive efficiency.

2-Decrease in appetite and food utilization so leads to decrease in the body weight in growing a normal & productivity in adult.

3-Lameness which is more common in the forelegs accompanied in young animals by bending of long bone and enlargement of the joints. (Rickets in young animals and osteomalicia in adult).

4-Decrease the fertility specially if phosphorous intake is low.

5-Dyspnoea if chest bone affected.

6-Arching of back. 7-Curvature of long bone.

Diagnosis:

1-History and clinical signs.

2-Laboratory diagnosis:

- low calcium and phosphorous level.
- low level of vit D
- High level of alkalin phosphatase 5.7units (N-2.5)
- Fecal analysis → GI. Parasitism.

Treatment and control:

1-Administration of vit.D R1-D-Varol ampl 2-7Iu/kg Bwt/day.

2-Supplementation of calcium and phosphorous in the diet.

3-Exposure to the sun light.

4-Fish liver oil is good source of vit-D (4,5)

5-Sun-dried hay is good source of vit-D2

6-Irradiated dry yeast is probably the simplest and cheaper method of supplying vitamin-D in mixed grain feeds.

7-In a mature non-pregnant sheep weighting about 50kg a single intera muscular injection at a dose of 6000 Iu/kg body weight produced concentration of vit-D3 at adequate level for 3 months.

8-The status of vit-D in lamb can be increased by parenteral injection of pregnant ewe 2 months before laming with 300.000 I.u. will provide a safe means of increasing the vitamin-D status of ewe and new born lambs by

preventing the seasonally low concentration of 1.25 dihydroxy vitamin-D3

9-Over dose may leads to vit-D toxicity

- *Drowsness*
- *Muscular weakness*
- *Fragility of bone*
- *Calcifecation of wall of bl.vs.*

*This occurs in unthrifty lambs receiving single dose 1million units, although larger doses are telerated by healthy lambs.

10-Oral dose 30-45 I.u/kg wt daily, massive single dose orally can be given and give long-term effect e.g: single dose of 2 millions units is an effective for 2 months in lambs.

5-Vitamin E and/or Selenium Deficiency

Synonyms:

1-Wh*te muscle disease "WMD" 2-Nutritional muscular dystrophy

3-Enzootic muscular dystrophy 4-Stiff lamb disease .

5-Selenium Respensive unthriftness "Se R.U"

Definition:

Complex syndrome of young lambs and kids due to deficiency of vitamin E and selenium during gestation period or early life and it's characterized by:

1-Acute or subaduct pathological degeneration and necrosis of skeletal and cardiac muscle and referred to as ***"white muscle disease"***.

"Nutritional muscular dystrophy NMD"

2-Sub-clinical cases characterized by poor growth and reproductively

and referred to as “selenium responsive unthrifthness (se R.U)”.

Etiology and pathogenesis:

1-Feeding on selenium deficient diet or net contain less than 0.2 p.p.m. or mg/kg Dry matter diet .

2-Feeding on inferior (bad) quality hay or roat crops or straw which usually deficient in selenium.

3-Feeding on diet contain excessive quantity of poly unsaturated fatty acids “i.e. rancidity which leads to destruction of vitamin-E”

Pathogenesis

1-Selenium is an essential component of glutathion preoxidase enzyme system “GSH-Px” in the erythrocyte.

2-“G.SH.Px” together with tocopherol (vit-E) and other enzymes as superoxide dismutase (S.oD) and Glutathion-S-transferase. (GSH-Tr) all these enzyme play an important role in protecting of mammaline cells from oxidante damage. So deficiency in vitamin E. and/or selenium leading to increase the oxidante damage of mammalin cell and degeneration of muscles.

3-Vitamin E-and selenium are separate nutrietns with simillar biochemical roles (anti-oxide ante).

4-Selenium playes an importante role in thyroid hormon which responsible for basal metabolic rate and growth rate and it act as thermoregulatory hormon as selenium enter in the structur of 5-deiodinase enzyme which responsible for conversion of the thyroid hormon

from in active form(T4) to the active form(T3) so selenium deficeicney leads to low function of 5-deiodinase enzyme and low level of active form of thyroid hormon resulting in low growth rate or thermal disturbances as hypothermia sepcailly in newly born animals.

5- Together with muscular exercise and vit-E selenium deficiency it lead to formation of H_2O_2 (hydrogen peroxid) which lead to:

A)-Hyaline degeneration

B)-Precipitation of calcium

C)-Pain on palpation of muscle.

D)-Caogulative necrosis.

E)-White muscle disease

F)-Lameness

Occurrence:

1-Occurs in all breeds, all sexes of lambs and kids from birth to 3 months of age but 2-4 weeks are common however the lesions development.

2-More common in lambs and kids whose dams subsist during pregnancy on alfa alfa or clover. i.e. “usually during spring and summer seasons”

Selenium in the soil

In Soil

Absorbed by plant and combined with selenate or selenide sulfure containing amino acid. As cystein, cystine or methionine.

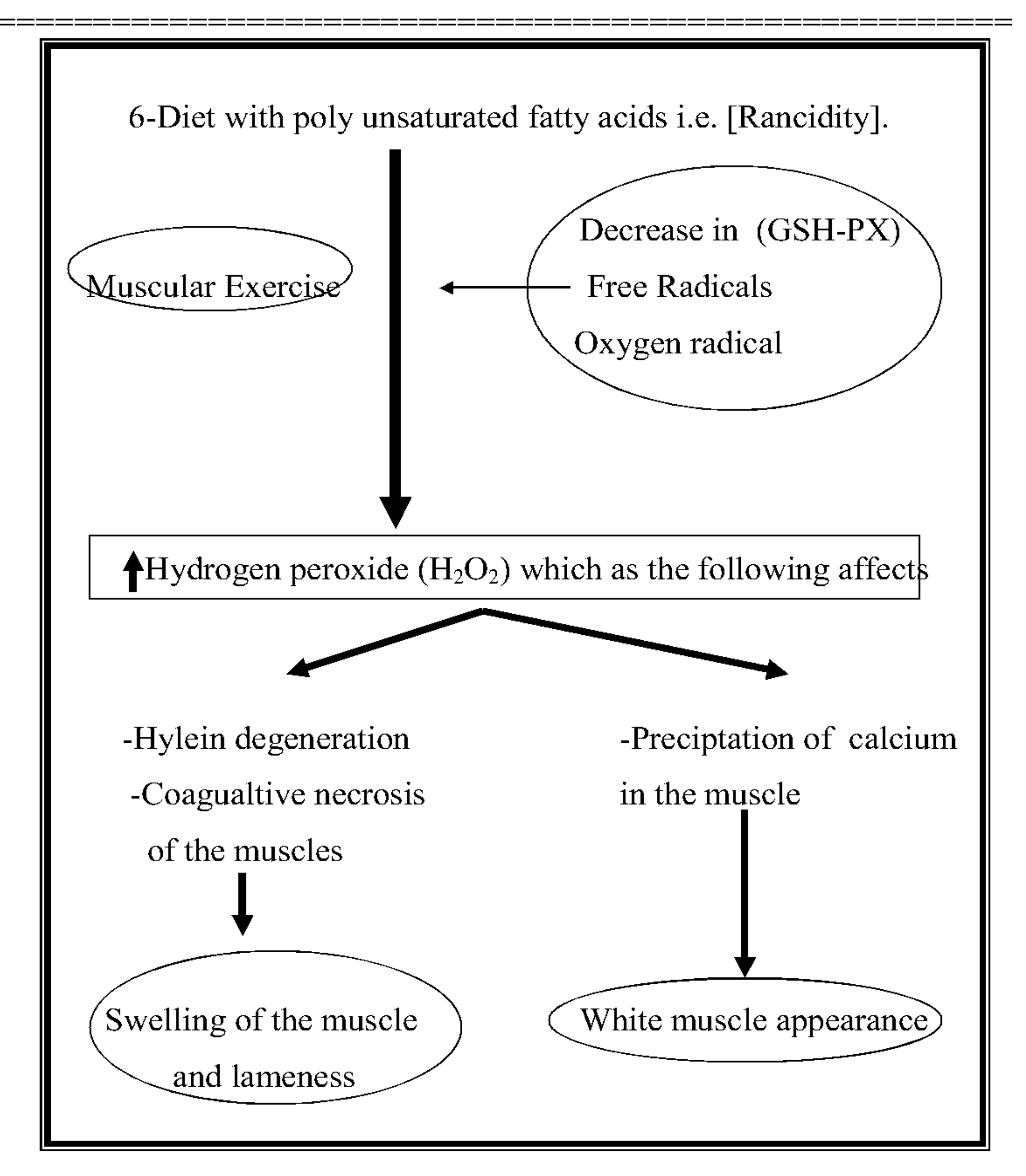
Selenioamino acids as
Selenoprotein in rumen as Selenocysteine

-Selenium is taken by the animal and destroyed by micro-organisms which present in the rumen to give selenioprotein(selenioamino acid).

-Selenoprotein reach the blood, placenta and milk to give (GSH-Px) Glutathion peroxidase enzyme [selenium + sulpher containing amino acid]

So low selenium intake → low level of GSH.-PX

Accumulation of peroxid and other oxidant agents that leadingto oxidative and damage of the cell causing the disease.



Clinical signs:

A)-Skeletal muscular dystrophy "sub-acute form"

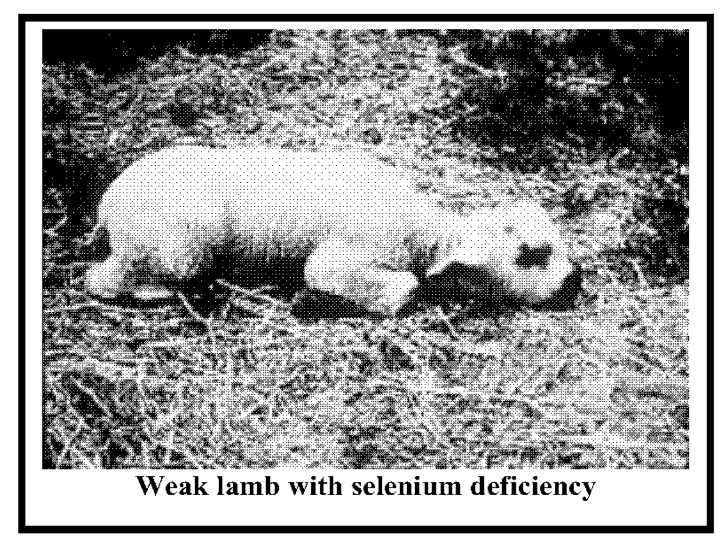
B)-Myocardial dystrophy "acute form "

A)-Skeletal muscular dystrophy "sub-acute form"

1-May appears at birth or 2-4 weeks of age.

2-Affected lambs show locomotor weakness and unsteadiness.

3-Because of muscular weakness the lambs prefer the reclining position.



Weak lamb with selenium deficiency

4-The animal may die without previous signs within 6-12 hours.

5-Shallow rapid respiration with frothy blood-stdined nasal discharge.

6-Rapid heart rate (Tachycardia) and irregular rhythm.

7-The temperature within normal range.

8-Goose strepping gait.

9-Sternal recumbancy.

10-Knucking at fetlock and standing on to relaxation of carpal and metararpol joints

11-Firm and swollen muscles, (shoulder & gluteal muscles).

12-Dyspnea if diaghragmatic muscles are affected.

B)-Myocardial dystrophy "acute form"

1-Weak pulse

2-Pulmonary odema

3-Lateral recumbancy

4-Accelerated breathing

5-Dyspnoea and death

6-Sudden onset of dullness

7-Affected animal commonly die within 6-12 hours.

8-The animal may die without previous signs.

N.B.

*GSH.Px: playes an important role in the regulation of action of G6pD [Glucose 6- Phsophate dehydrogenase Enzyme] which is very essential for maintaining of RBCs fragility and consequently deficiency vit-E & selenum causing defecte in GSH-Px and subsequently followed deficiency in G6-PD enzyme causing haemslysis of RBCs and anemia what so called favism.

*GSH.Px → protect the cell wall or membrane from destrcution by oxidant agent as H_2O_2 by stabilizing the fat contents of the cell membrane.

Diagnosis:

I)-History and clinical signs.

II)-PM lsions: usually concerned with skeletal and cardiac muscles

A)-White to pale lesions of muscles including inter costal muscles.

B)-Presence of linear white streaks due to affected fibers bundles within the muscles.

C)-Peticheal haemorrhage and muscular adema may occurs.

D)-In cardiac muscles the same lesions present but concerned with sub-

endocardial muscles and may extended to the septum.

E)-If death due to left ventricular failure it leads to cengested and odematous lungs. I.e. pulmandany edema.

III)-Histopathologically *:--Hyaline degeneration and coagulative necrosis of the muscles*

III)- Differential diagnosis:-

With sway back disease or enzootic ataxia in which the stiffness is not prominent as in white muscle disease.

Laboratory diagnosis:

1-Estimation of muscles specific enzymes.

A)-Cretine phosphokinase enzyme (CPK) specific for muscular degeneration, usually 1000 I.u/liter but commonly as high as 5000-10.000I.u/l (N=25-65I.u/liter).

B)-Serum glutamic oxal acetic transaminas or AST. (sGot) but not specific in muscular degeneration as "CPK", it up to 300-900 I.u/liter (N= up to 100).

2-Determination of vitamin E status (tocopherol):

*Highly expensive

3-Determination of selenium status either in the soil,plasma or in the animal tissus

*Difficult and highly expensive (N=0.05-0.06 PPM)

4-Determination of "GSH-PX"

-Deficient level → 30 millimicrone/mg Haemoglobine

==
-Marginal level→ 30-60 millimicrone/mg Haemoglobine

-Adequate level is greater than → 60 millimicrone /mg Haemoglobine

Treatment: 1-R/ Sodium or potassium selenite 3mg.

+ alpha tocopherol acetate 150I.u. + water

taken I.M. single dose (2ml/45kg Bwt.)

2-R/ Vetacllin inj. up to 10ml I.M.

3-R/ Alpha seline inj. up to 10ml I.M.

4-R/ Vit-E & selenium. Powder or solution.

5-The normal dose of sodium or barium selnite is 0.03 mg/Kg Bwt.

Control:-

1-Feeding on diet or ration adequate in vitamin-E and selenium and free of polyunsatuated fatty acids.

2-Barium selenit → if it given to pregnante ewes it can maintain the level of selenium of lambs up to 6 months as it can pass through the placental barrier.

3-Monensin as feed additive has been shown to elevate GSH-PX activity without an increase in dietary selenium (monsesin used for controlling of coccidiasis in lambs as feed additive).

4-Maintaning of the normal level of selenium in the diet, not less than 0.3ppm. of DM.

6- Zinc Deficiency

Synonyms: 1-Wool eating disease 2- Parakeratosis.

Definition:- On of most important nutritional deficiency in sheep and goat due to zinc deficiency characterized by wool eating, parakeratosis, alopecia, lameness, unthriftness, abnormal hoof and poor growth.

Etiology:- ***1-Primary*** due to feeding on zinc deficient diet, also the soil of PH. Above 6.5 and contain high amount of phsophorous and Nitrogenous fertilize is deficient in zinc, so it's producing plants is deficient in zinc also.

2-Secondary due to:

A)-Consumption of immature grass which affect the digestibility.

B)-Feeding on late cute hay which may be poorly digestible and contain of excessive dietary sulfur which hindrance the absorption of zinc.

C)-Silage which contaminated with soil also affect the digest ability of zinc.

Pathogenesis:- Not completely understood"

1-Zinc is component of carbonic anhydrase enzyme which present in RBCs and glandular cells of stomach ,so such enzyme is important for (Transportation of respiratory carbone dioxide and release and secretion of gastric juice "Hcl").

2-Zinc usually associated with RNA function, glucagon, insulin and other hormones.

3-Plays an important role in healing of the wound, keratinization of the

skin, calcification of bone, and development of somatic and sexual cells. i.e.. spermatogenesis.

4-Has critical role in protein and nuclec acids metabolism, so it's deficiency leads to defect in cell mediated immune system.

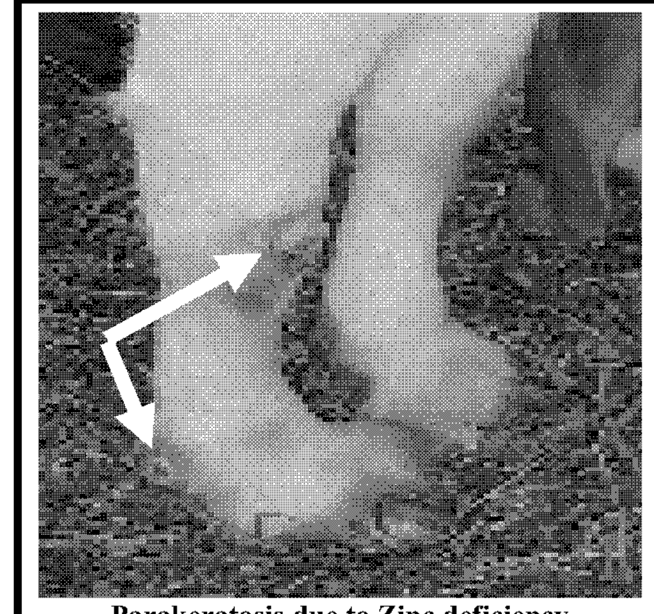
*As we mention the role of zinc and it's importance so it's deficiency leading to disturbance in all mentioned process and manifested by the following clinical signs.

Clinical Symptoms:

1-Alopecia (loss of wool in sheep and hair in goat with rough hair).

2-Development of thick wrinkled skin (parakeratosis).

3-Wool eating as first signs specially in lambs after deficiency for 4 weeks.



Parakeratosis due to Zinc deficiency

4-Poor growth rate, swollen hocks and excessive salivation specially during rumination. 5-Opened skin lesions around the eyes and hoof.

6-Decrease the fertility in rames and cessation of spermatogenesis.

7-Arched back and abduction of legs during standing position.

8-Low food intake (Anorexia). 9-Fissured hoof with exudate or

sluphing of it. 10-Animal become highly susceptible to be infected with infectious diseases.

Diagnosis: 1- History and clinical symptoms.

2-Differential diagnosis with mange in which → more itching and rubbing. And skin scraping test show presence of the parasites.

3-Laboratory diagnosis: *Skin biopsy → parakeratosis.

*Zinc level reach 18µg/dl (N=80-120µg/dl) but level below 39µg/dl considered as zinc deficiency.

Treatment: 1-Zinc sulfate or carbonate 200mg/kg of food.

2-Calcium should be maintained at level 0.65 to 0.75% of diet not more because excessive calcium decrease the absorption of zinc.

3-Injection of zinc at level of 2-4mg/kg Bwt. For 10 days is good.

4-Zinc oxide suspended in olive oil and injected IM 200mg of zinc oxide is sufficient to ewe and 50mg for lamb give good result within 2 months.

5-Oral administration of zinc sulfate 250mg/day for 4 weeks gives cure in goat in 12-14 weeks.

Control: 1-Interarumenal pellets give protection for 7 weeks only.

2-S/c. Depots by injection of zinc oxide s/c.

3-Supplementation of zinc in diet.

4-Avoiding feeding on plants from soil deficient in zinc.

5-Avoid excessive sulfer or calcium in diet.

6-Feeding on good digestible food.

7-Copper Deficiency

Synenomes

1- Enzyootic ataxia 2-Sway back disease

Definition:

It's one of important metabolic disease due to copper deficiency in lambs and kids up to 3 months of age characterized clinically by progressive in coordination of hind limbs and pathologically by disruption of neurons and myelin development in the central nervous system.

Occurrence:

(1)-Usually in lambs up to 3 months of age due to copper deficiency in the last half of pregnancy of dame

(2)-Occurs in most sheep and goat producing countries.

Etiology:

1-Mainly due to deficiency of metabolisable copper in pregnant animals.

2-Primary deficiency mainly due to soil deficient copper, so feeding plants grown on such soil leads to enzootic ataxia or Hypocoprosis.

3-Secondary copper deficiency: occurs when copper intake in the diet is sufficient but the utilization of copper by tissues is impeded due to dietary excess of Molybdenum and in organic sulfate alone. Or in combination of organic sulfat and molybdeum or cadmium.

4-Molybdenum leads to increase excretion of copper and decrease it's absorption from intestine and, it's storage in the liver.

Pathogenesis:

If the copper level:-

1-In plasma below 50ug/100ml (N=70-120)-In liver below 80 mg/kg dry matter (N = 120-1000) considered as copper deficiency and leads to Enzyootic ataxia.

2-Copper play an important role in "tissue oxidation" by formation of copper-containing enzymes as :-

-Cytochrome oxidase

-Ceruloplasmin (copper-protein complex)

-Superoxide dismutase (antioxidante)

-Tyrosine oxidase

- lysyle oxidase.

These enzymes are essential for:

1-Controlling the phagocytosing efficiency of leucocytes.

2-Normal oxidative phosphorylation and tissue oxidation.

3-Control over inflammatory response.

4-Protection of mylein sheath from oxidation by the oxygen radical [hydrogen peroxid H_2O_2].

5-Neutralization of free thiol group which essential for keratinization of wool in sheep and hair in goat

6-Copper is essential for neutralization of iron for forming of haemoglobuin.

7-Elastine and collagen maturation by lysyle oxidase enzyme.

8-Clearance of plasma from cholesterol.

9-Production of mylein sheath [by cytochrome oxidase enzyme]

10-Metabolism and growth of the animal through oxidation, through the action of the Tyrosing oxidase enzyme .

So deficiency of copper leading to depression of such enzymes that leading to disturbances in it's functions and this disturbance manifested in the form of clinical signs .

Clinical Signs: ***(A)-General symptoms:***

1-Crimpless wool and general unthriftness and steely appearance of the wool.

2-Anaemia and depigmentation of black wool or gray white.

3-Course varies from few days to several weeks.

4-Morbidity up to 90% with high mortality rate among affected animals.

5-Diarrhoea (scouring) and osteoperosis in extreme deficiency case.

6-Steely wool and anemia.

(B)-Sawy Back and Enzootic Ataxia :-

Sumariezed in the following table in the following page.

Diagnosis:*(I)-History and clinical signs.*

(II)-Laboratory diagnosis:

(A)-Plasma copper is below 50ug/100ml (N=70 –120mg/100ml)

(B)-Liver copper level is below 80mg/kg dry matter (N = 120-1000 mg).

(C)-Low copper content of hair (goat) (N = 6.6 – 10.4mg/kg dry matter).

(D)-Estimation of Erythrocyte superoxide dismutase enzyme 2-6 I.u/mg haemoglobine in case of Hypocuprosis.

(E)-Estimation of ceruloplasmin (N = 40–100 gm/dl)

A- Sway back	**B-Enzootic ataxia**
(1)-Congenital form :- (cerbrospinal swayback) the affected lambs born dead or weak anable to stand or suck ***(2)-Delay form*** (progressive spinal sway back) usually 3-6 weeks of age (develop some week after birth). ***(3)-Postnatal form*** [acute fatal, sway back) similar to delayed form but developing suddenly and lead to death within 1-2 day due to acute swelling of cerebrum. (4)-The signs of 3 forms are: * In coordiantion, paralysis and blindness.	(1)-Affect only unweaned lambs. (2)-Most cases at 1-2 months of age. (3)-Less incidence at birth. (4)-Lambs affected at birth usually die within 3-4 days while older lambs may last 3-4 weeks. (5)-In coordination of hind limbs due to parasis not paralysis. (6)-Felxion of joints. (7)-Knuckling over the fetlocks. (8)-Wobbling of hind quarters and finally falling. (9)-Recumbancy when the forlegs become involved.

Treatment:

1-1gm of copper sulphate for lamb orally for 3-5 weeks (weekly)

2-2-3gm copper sulphate for adult orally.

3-Single parental injection of copper glycinate 150mg IM or s/c

4-Mineral mixture contain at least 3-5% of copper sulphate.

Prevention:

(1)-Supplementation of diet with copper sulfate 5mg copper per kg of matter of food by any of several different ways:

(2)-oral dosing or dietary supplementation 1.5gm for sheep/week.

(3)-Mineral mixture of salt licks containing ¼ - ½% copper sulfate.

(4)-Parenteral injection of (Copper glycinate/copper methionate “150mg” IM or s/c /copper calciume edibate).

(5)-Single injection 45mg of copper glycinate in mid pregnancy is sufficient in prevent sway back in lambs.

(6)-Copper oxide needles:

- Fragments of oxidized copper wire 8mm in length and ½ mm in diameter for oral dosing considered one of the most effective and safest methods for control of copper deficiency in ruminants.

-The needles retained in fore stomach and abomasum for 1000 days or more and copper slowly released, absorbed, and stored in the liver.

8-"Hypovitaminosis-A"

Definition:- Hypovitaminosis-A means that deficiency of vitamin A due to deficient diet of vitamin-A (Primary cause) or imperfect digestion, absorption or metabolism of vitamin-A and It's characterized by affections of CNS. In young animals or night blindness, pitryiasis, corneal keratinization loss of body weight and infertility in adult animal.

Etiology:

(1)-Primary cause: Due to inadequate supplementation of vitamin-A in the diet of Feeding on vitamin-A deficient diet.

(2) Secondary:- Due to imperfect digestion, absorption or metabolism of the vitamin due to disease of the liver or intestine although the supplementation of the vitamin in the diet is adequate.

Epidemiology:

(A)-Primary deficiency:-

(1)-Mainly due to lack in green food which is rich in vitamin-A.

(2)-Vitamin-A present in two forms which are alcoholic form. (caroten or precursor of vitamin-A)and ester form (synthetic parental preparation).

(3)-Alcoholic form of vitamin- (caroten) not pass through the placental barrier so if the dame depend upon green fooder in late stage of pregnancy that not leads to increase the level of the vitamin while ester form of vitamin-A which present in commercial preparation and fish oil has the ability to pass through the placental barrier, so giving of

such products in late pregnancy of the dam leads to increase the hepatic storage of vitamin-A in lambs or kids.

(4)-Taking of vitamin-A by any form increase it's concentration in the colostrum which decrease within few days after parturition so lack of colostrum in the first 48 hours after parturition leads to hypo vitamins-A of the newly bome animals (lambs or kids).

(5)-Vit-A is fat solubal vitamin, so it's rapidly oxidized in presence of unsaturated fatty acids or a mineral oil as parafin oil which may be used in treatment of bloat or impaction, so the oilly form of the vitamin is not-satesfactory as other form-of the vitamin-A specially storage for long period.

(6)-High temperature, light or Mineral mixture also increase the destruction of vitamin-A in the commercial ration.

(7)-Hepatic storage in young animal may extend up to 1 years while hepatic storage in adult animal may extend up to 1½ year.

(8)-Grains with exception of the corn grain are deficient in vit. A also cereal hay are poor source of vitamin-A.

(B) Secondary deficiency:

(1)-Mainly due to diseases of the liver or intestine that due to most of the caroten (precursor of vitamin-A) is converted in the intestinal epithelium and the liver is the main site of storage of the vitamin A.

(2)-Increase the level of inorganic phosphorous causes decrease the storage of vitamin-A in the liver But low level of phosphorous may lower the conversion of caroten into vitamin-A.

(3)-Vitamin-E and C help in prevention of the loss of vitamin-A from the food stuffs by their anti-oxidant action.

(4)-High temperature and nitrate decrease the conversion of caroten into vit.A

(5)-Highly chalorenated naphalene substances cause low level of conversion of the caroten into vit-A so poisoned animal by such substances suffering from low level of vit-A status.

Pathogenesis:- Vitamin-A play an important role in:

(A) Night vision (B) Cerebrospinal fluid pressure: (C.S.F):

(C) Bone growth (D) Epithelial tissues:

(E) Fetal tissues (F) Immune mechanisms:

(G) Other abnormalities:

*So vitamin-A deficiency leads to disturbance in such process and the most pathophysic logical effects of vitamin-A deficiency are as follows:

(A) Night vision:

-The ability to see in dim light is reduced due to interference with regeneration of the visual purple.

(B) Cerebrospinal fluid pressure: (C.S.F):

1-An increase in C.S.F pressure is one of the first abnormalities to occur in hypo vitaminosis-A in calves,lambs and kids.

2-It's a more sensitive indicator to hypo vitaminosis-A than ocular changes and in the calf it occur when vitamin-A intake is about twice that

needed to prevent night blindness.

3-The increase of C.S.F pressure is due to

a) Reduce tissue permeability of the arachnoid villi.

b) Thickening of the connective tissue matrix of the cerebral dura matter.

4-Increase of C.S.F pressure is responsible for:

* Syncop *Convulsion that occur in small animal

5-Convulsions and syncope may occur spontaneously or be precipitated by excitement or exercise.

6-C.S.F. pressure may be increased in sub-clinical cases. And exercise may evoke it to convulsive levels.

(C) Bone growth:

1-Vitamin-A is essential for maintaining of normal position of the osteoblasts and osteoclasts cells, so vit-A deficiency leads to No-retardation, of the endocondral bone growth but there is in-coordination of the bone growth.

2-Vit-A deficiency lead to facial paralysis and blindness due to constriction of the bone canal through which the facial and optical nerve passe.

3-Vit-A deficiency leads to decrease the size of skull causing pressure on brain causing nervous manifestation as syncope and convulsions due to increase of the C.S.F. pressure.

(D) Epithelial tissues:

1- Vit-A is essential for all type of epithelial cells, so vit-A deficiency lead to atrophy of epithelial tissues specially glandular cells as well as epith tissues which have covering function [as skin].

2-The glandular epithelial cells are gradually replaced by keratinized or stratified non-secretory cells as in salivary gland, intestinal cells, paraorpital gland or thyroid gland………etc and placenta.

3-The most important changes that occur due to epithelial changes including placental degeneration, enteritis, xerophthalmia and dermal changes.

(E) Fetal tissues:1-Vit-A is very essential for developing of all organs of the fetus.

2-Vit-A deficiency in pregnant dame may lead to congenital hypovitaminosis-A of the lambs, kids or calves which characterized by:

A)-Blindness due to constriction of the optic canal with the optic nerve and optic disc edema wich leads to blindness.

B)-Hydrocephallus. C)-Dilated pupils

D)-Nystegmous E)-Weakness and in coordination.

(F) Immune mechanisms:-

1-Experimentally sever vitamin-A deficiency in calves lead to alteration in the immune function but the exact mechanism in unknown..

2-Some workers claim that the vitamin-A may protect the animals from infection by bacteria, viral, rikettsia or parasite as it influence both specific and non-specific host defense mechanisms, so vit-A deficiency

may associated with immune suppression that lead to the animal become highly susceptible to infection by bacteria, virus…etc.

(G) Other abnormalities:

Vitamin-A deficiency has been associated with left side abomasal displacement and anasarca of the ventral abdomen in dairy heifer but the mechanisms until now is unknown.

Clinical Finding: it includes:

A-Night blindness	B- Xerophthalmia
C-Skin abnormalities	D-Reproductive in sufficiency
E-Nervous manifestations	F- Loss of body weight
G-Congenital defects	H-Other diseases:

A- Night blindness: In ability to see in dim light is the earliest sign.

B- Xero phthalmia:

**In calf*: there is true xerophthalmia with thickening and clouding of the cornea.

In other spp.: thin, serous mucoid lacremal discharge followed by corneal keratinization, clouding and sometimes ulceration of the cornea and photophobia.

C- Skin abnormalities:

1-Presence of bran-like scales on the skin with shaggy appearance.

2-Dry, scaly hooves with multiple, vertical cracks specially in horse.

D- Reproductive insufficiency:

1-In male: *Libido is retained. *Testicular degeneration

*Production of small numbers, and normal sperms [Azospermia]

2-In female: *Conception usually not affected.

*Placental degeneration which may lead to abortion.

*Retained placenta. *Dead or weak birth.

E- Nervous manifestations:

1-Paralysis of the skeletal muscles due to damage of peripheral nerve roots.

2-Encephalopathy due to increase in the intera cranial pressure.

3-Blindness due to constriction of the optic nerve canal.

4-Curvature of the spinal column with in coordination of gait.

5-Convulsive seizures which are common in beef calves at 6-8months.

6-Spontaneously or following exercise or handling the affected calves may be collapse (syncope) and during recumbancy clonic-tonic convulsion will occur lasting for 10-30 seconds, and may be die, during convulsion.

7-Menace reflex may be slightly impaired or hyperactive.

In calves during convulsion:

1-Ventero flexsion of the head and neck.

2-Opisthotonus and tetanic closure of the eye lids and retraction of the eye ball.

3-Out breaks of this form of hypo vitaminosis-A is recorded and cause fatality 25% but treatment gives good prognosis within 48hr, but the

convulsions may continue for up to 48hr, following treatment.

Ocular form of hypovitaminosis-A: manifested by

1-Fixed and dilated eye puple with not-response to light.

2-Usually in yearling calves and up to 2-3 years of age the animal cannot reach the vitamin.

3-The menace reflex is usually totally absent but the palpebral and corneal reflexes are present and the animal is aware of it's surrounding and usually eats and drinks unless placed in usnfamilliar surrounding.

4-Varying degrees of peripapillay retinal detachment, papillary and peripapillry retenal haemorrhage and disruption of the retinal pigment epithelium may also be present.

5-The prognosis for these is unfavorable and treatment is in effective due to degeneration of the optic nerve.

F- Loss of body weight:

1-Vitamin-A deficiency lead to abnormal metabolism of carbohydrate and patein leading to emaciation and loss of body weight [expermentaly]

2-But in most cases the the animal may maintain it's body weight in most cases of vitamin-A deficiency.

G- Cngenital defects: [congental hypovtaminosis-A].

1-Congenital blindness due to constriction of optic nerve.

2-Hydrocephellus and encephalpathy.

3-Dilated pupils nystegmus and in coordination and weakness.

H- Other diseases:-Enteristis and otitis media-Anasarca and abomasal displacement.

Diagnosis:-1-History and clinical finding.

2-Laboratory diagnosis:

A) Estimation of liver and plasma vitamin-A :-

1-Plasma carotin vary largely with diet [level about 150ug/dl is optimum]. 2-Plasma vitamin-A concentration ranged fram 25-60ug/dl.

3-Hepatic level of → Carotin ½-4 ug/gm liver/vitamin-A 2-60ug/gm liver.

B) Estimation of C.S.F. pressure (Normal less than 100mm of water , abnormal pressureis more than 200mm of water).

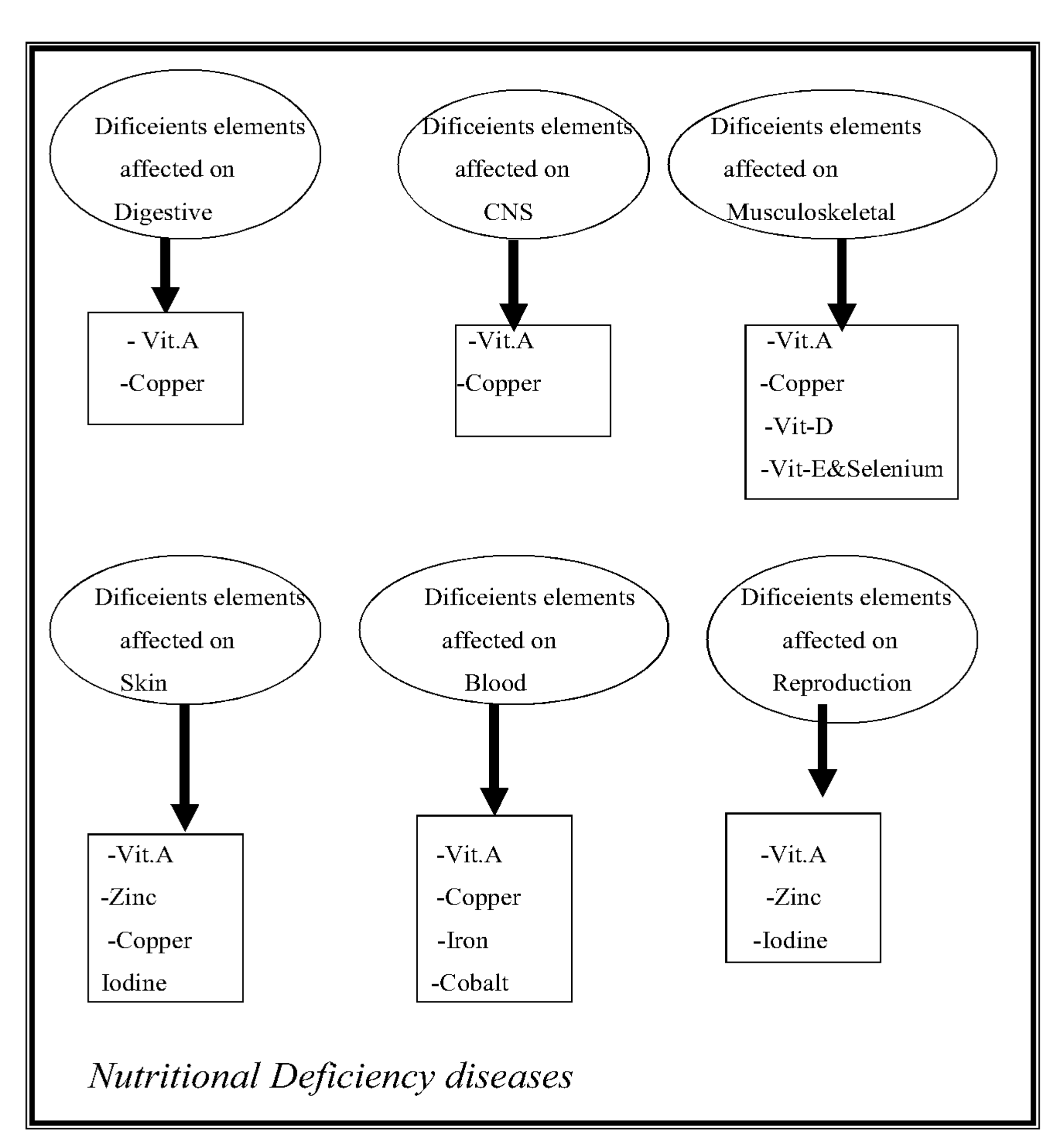
C) Conjuntival smears: we found increased number of cornified epsthelium cells.

Treatment:-1-Supplementation of vitamin-A within about 1-20 times of that of normal requirements of the animal which is 40 I.u/kg Bwt.

Control:-2-Providing the daily maintenance requirement of vitamin-A which not less than 40I.u/kg/Bwt, daily and it msut be provided during pregnancy.

3-I.M. injection of 3000-6000 I.u/kg Bwt, vitamin-A within intervales of 2months gives optimum plasma and hepatic level of vitamin-A

N.B in pregnancy 2 doses [at 6 and 8^{th} months] give good result and it's prefered to give the last dose to the ewe at one month before parturition.



Nutritional Deficiency diseases

Chapter No.3

Digestive System

1-Dental attrition 2-Periodentitis

3-Chok 4-Bloat

5-Ruminitis

6-Ruminal parakeratosis

7-Abomasal ulcer

8-Impaction 9-Enteritis

1-"Dental Attrition"or "Tooth wear"

Definition: Dental attrition means that rapid and excessive wear of teeth characterized by inefficient prehension and mastication, and utilization of feed caused by abrasion of soft teeth by diets.

Economic losses due to:1-Inefficient use of feed.

2-Premature culling of affected animals. 3-Lower production .

Occurrence: 1-Dental attrition occurs in all types of sheep and goats.

2-Ewes 5 years old and old aged animals are highely susceptible than do other categories of sheep and goats.

3-Geographically in all sheep producing countries spcaially that own sandy pasture or arid and semiarid soil.

Etiology and pathogenesis:

1-Mastication of sand and soil along with prehended forage causes dental attrition.

2-Calcium deficiencies with softness of tooth enamel and dentine may accelerate the rate of wear of the teeth.

3-Malalignament may predispose individual teeth to excessive wear.

4-Pathogenesis: of dental attrition usually related to the animal and the diet as: *(A)-If the animal exposed to calcium deficiency or in balanced ratio between calcium and phosphorous specially in 1st. 18month it leads to softness of tooth enamel and dentine.*

(B)-Ewes supporting annual pregnancies lose dental calcium.

(C)-During grazing specially short forage the animal ingest and masticate soil and sand which with long time it leads to wearing of ncisor and molar teeth.

(D)-The animal affected become malnourished and die from exhusation or inter current diseases.

Clinical Signs: 1-Abnormal prehension and mastication.

2-Slaivation , weakness and unthrifthness.

3-Emaciation and anemia. 4-Death due stravation, pneumenia or pregnancy disease.

5-***PM***: weared teeth or erosions on the gum.

Diagnosis:

1-History and clinical signs. 2-Deferential diagnosis with periodental disease and chronic progressive pneumonia.

Prevention and Treatment:

1-Calcium deficiencies should be avoided and treated by supplementation of balanced diet and injection of calcium borogluconat respectively.

2-1% ground lime stone in the ration supplies adequate calcium.

3-In the autumn, all sheep should be examined for unsound teeth, and all animals showing excessive dental wear should be culled and markated or maintained on pre-ground feed.

4-In winter supply sufficient feed to avoid grazing and reduce the amount of masticated soil or sand.

2- Perio Dental Disease "PD"

Synonyms:1-Periodentitis 2-Periodentosis 3-Cara inchada 4- Broken mouth

Definition:- It's chronic infection of the sockets of mandibular teeth characterized clinically by loosing, lengthening, wearing, spreading, malocclusion, and expulsion of an individual teeth and pathologically by destruction of the period dental ligments and osteopathy.

Economic loss due to: Unthriftness, inefficient use of food and low production of meat and wool and early culling of premature animals.

Occurrence:

1-PD occurs in all breeds, sexes and ages but more incidence of 3 years or older ewe than do other age groups.

2-Occurs in all seasons, prominent in New Zealand, USA, Scotland, and other sheep producing countries.

Etiology and pathogenesis:

1-Usually association between nutritional deficiency especially calcium together with bacterial infection as diphethroid or bacteroids gengivalis.

2-The perio-dental tissues (periodntium) consists of:

A-Epithelium of the gum. *B-Fibers of ligament (periodental ligament)*

C-Cementum of the tooth. *D-Bone of the alveolus.*

2-At the gengival sulcus, the epithelium normally attached to the enamal or cementum, so it covers the subjacent tissues.

3-The fibers of periodental ligament attached centrally to the comentum

and peripheally to the bone, suspende the tooth in the socket and absorb energy from occlusal pressure.

4-The initial pathological changes probably begin with degeneration of cementum, periodental fibers and bone or possibly with gengivitis.

5-Malcalculus attaches to the enamel at the gengival sulcus, so movement of the tooth and calculus leading to errosion and ulceration of epithelium that predispose and help the entrance of bacterial infection to the ligament leading to periodentitis.

6-Repetition of such process leading to formation of pocket 1-2ml deep all or part of the circumference of the affected teeth.

7-Pathological changes may reach the alveolus and bacterial infection may leading to absecess formation and in advanced case expulsion of the tooth occurs and the socket is filled with granulation tissues and covered over by epithelium.

8-The disease usually does not spread to other teeth.

9-Also acute gengivits → chronic gengivits.→ penetrate down into the alveoli causing the disease.

Clinical Signs:

1-Loss of incisors teeth usually between 3.5 to 6.6 years while healthy sheep retain it's teeth beyond 7 years of age.

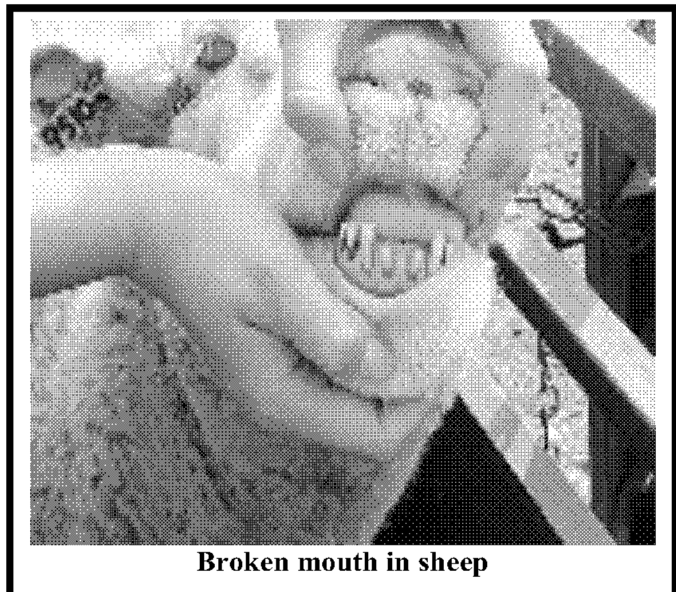
2- Teeth movement, gum recession and pocketing.

3-Gengivitis which characterized by redness and odema of attached gingiva.

4-Incisors and molar teeth usually affected.

5-Crown lengthening, protrusion, hemorrhage, loosening and lingual periodontitis are characteristic.

6-Unthreftness and starvation due to inability to fed with loss of body weight.



Broken mouth in sheep

7-Abscesse formation may be detected due to bacterial infection.

8-Digital pressure may express exudate around the tooth.

9-Difficulty in prehension and masticatin.

10-Salivation and dropping of fragments of food from mouth during the chewing movement.

11-Hypertrophy of the gum.

12-Incidence varies up to 10% and course extends through many months.

Diagnosis:- 1-History and clinical signs.

2-Laboratory examination by isolation of (spirochets and fuso bactrium common).

Prevention and Treatment: 1-Avoid nutritional deficiency.

2-Providing of mineral mixture. 3-Antibiotics to over come the bacterial infection.

4- Oesophegeal Obstruction

Synonymes:1-Esophegeal occlusion 2-Choke

Definition:- Choke is the accidental lodgment of the oesophegus by ingesta or foreign body, and such obstruction may be partial or complete.

Occurrence:- 1-Occurs in all breeds, sexes and more common in 4-6 months of age specially in feed lot lambs and kids.

2-More common in late summer and early autumn especially the weaned lambs and kids grazing harvisted fields commonly develop obstruction.

Etiology and pathogenesis: 1-Eating globular food which has a size or diameter larger than that of the oesophegus, so leading to lodging of such material in the eosophagus causing oesophegeal obstruction.

2-The foods similar to corn cups, sugar-beet crowns, suger beets fruits, potatoes and carrots are most commonly incirmenated in choke.

3-When the foreign body or globular food which has larger diameter than that of the oesophegus lodged in the oesophegus it leads to:

a-Pressure on the wall of oesophegus cousing it's obstruction.

b-Accummulation of saliva in the eosophagus over to the site of the obstruction.

c-Prevent the eructation, so leading to accummulation of gases in the for stomachs and bloat or tempamy [free gase temary].

d-Saliva is regurgetated from mouth and nostriles and some of it may enter the larynx and trachea which may leading to aspiration pneumonia

or drenching pneumonia.

4-The obstruction in sheep usually occurs in the cervical part of the oesophegus but it may be occur in the thoracic part near to the cardia of the stomach.

5-If the animal neglected it will be die from pneumonia ,bloat or asphyxia.

6-Oesophegeal obstruction may occurs due to external pressure on the oesophegus as in case of neoplasmes or enlargment of mediastinal lymph node or carcenoma of stomach may leads to obstruction of the cardia.

7-Pneumonia may be associated with the singes of the eosophegeal obstruction if it is associated with enlargement in the mediastinal lymph node.

Clinical Signs:- 1-The onset of the disease is sudden and the animal is restlessness and alarmed.

2-Strong and repetitive movements of swallowing and mastication (dysphagia). 3-Extension of the head and neck.

4-Regurgitation of food immediately after swallowing, and if food only is regurgitated this indication to incomplete oesophegeal obstruction. While if water and food is regurgitated this indication to complete oesophegeal obstruction (bad prognosis).

5-If the obstruction near the larynx it leads to cough and dyspnoea.

6-Bloat is developed rapidly after obstruction.

7-If the lodged material present in the cervical part of the oeseophegus, it

can be palpated (gentelly) from left side [side of examination of the oesophegus].

8-If the animal is neglected it will be die due to pneumonia, asphyxia and bloat.

PM:1-Lodged food particles or foreign body in the oesophegus.

2-Necrosis of oesophegeal wall at the seat of obstruction.

3-Trachea may contain aspirated fluid and pneumonia.

4-Tympanic animal.

Diagnosis:- 1-History and clinical signs & PM lesions.

2-Confermatory diagnosis by:

a-Passing of stomach tube (Passe in incomplete obstruction and not passe in complete obstruction)

b-Palpation of osophegus at left side .c-Free pharynx.

***Treatment:-Hygienic treatment*:**

*Prevent food intake completely and excessvie water intake or lubricating mineal oil).

A) ***Supportive treatment*:** -Parenteral feed (Dextrose 5% I.V).

B) ***Medical treatment:***1-Give tranquilizer to calmming the animals as R/Rombun or Neurazing or cholaral hydate orally 5%

2-Compete the bloat by trocar and canula or large gauge needle.

3-In case of cervical part obstruction we can make pushing the lodged particle by gental massage of oesophegus for ward and up ward or take up

it by long forceps.

4-In case of obstruction in the thoracic part of the oesophegus we can push it by stomach tube toward rumen.

D) ***Surgically treatment*** :- By oesophegotomy or rumentomy.

4- Bloat or Tympany or Typmpanites

Definition:- Bloat is a retention of gas in the rumen and characterized by increase the interaabdominal and interathoracic pressures and caused by complex interactions of plant animal and microbial factors.

Economic losses due to: Death, unthriftness, high costs of theapeutic and prevention and resarch and incomplete utilizatin of bloat-provoking feeds.

Types of bloat: ***(A) Froathy bloat due to either*:**

1- Green legumes (green legum bloat GLB)
2- Hay legumes (Hay legumes bloat H.L.B)
3- Grain concenterate (Grain concenterate bloat G.C.B)

(B) Free gas tempany.

Occurance:- 1)-Occurs in all breeds, all sexes of lambs from birth to 3 months of age but 2-4 weeks are common however the lesions development.

2)-More common in lambs whose dams subsist during pregnancy on alfa alfa or clover. i.e. "usually during spring and summer seasons"

3)-All categories of bloat occurs in all breeds, sexes and ages beyond weaning age.

4)-GLB is high in lambs 3 to 9 months specially when transported from range to agricultural pasture of sacculant alfa alfa or clover for growth and fattening.

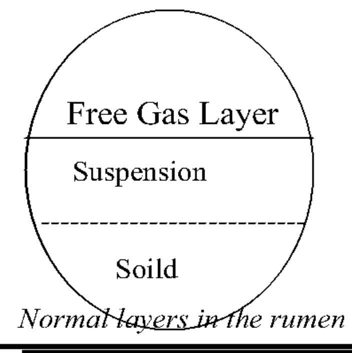
4)-Normally the lambs fed on graze legume pasture for 1-4 ws before entering feed lots to complete fattening so hunger lambs eat rapidly and excessively and become bloated (Grain concentrated bleat GCB).

5)-Seasonally: *GLB → during plant growing season i.e. sping season. *H.L.B → during all sesons. *G.C.B. → in late summer and early fall following weaning in feed let lambs.

6)-Geographically → in all sheep producing counteries.

Etiology and pathogenesis:

Normally:



Normal layers in the rumen

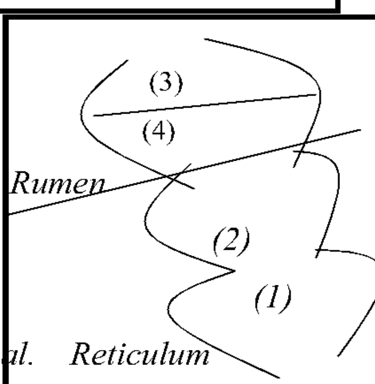
1)-Gas production in the rumen play role in:

**Aerobic fermentation.*

**Neutrilization of salivary bicarbonate.*

2)-Gases are: Co_2 45-75%. , Methan 20-30%and N_2, O_2, H_2 and H_2S (minor amounts)

3)-Gases are eleminated by eructation by contraction of dorsal rumenal sac and in normal animal the reticulorumenal movements consists of:



A)-Biphasic contraction of the reticulum 1,2.

B)-Monophasic contraction of dorsal rumenal. Sac "3" followed by contraction of venteral rumenal. Sac "4" [Primary Rumenal Cycle]

4)-[Secondary cycle] followed by alternated primary cycles and consists of monophasic contraction of the dorsal ruminal sac and then venteral rumenal sac and the frequency of secondary cycles are variable and related to the animal's need to eructate, according to amount of
gases that coallose in free layer.
5)-Gaseous distension in the rumenoreticulum leads to increase the force and frequency of secondary cycles,stimulate the opening of the caudal eosphegeal sphineter, and initiate the respiratory, oesophegeal, pharyngeal and buccal action of eructation sequences.
6)-Oesophgeal sphineter is open "reflexly" when dorsal rumenal sac contract in the secodnary cycl leading to (Forcing the gases layer forward and depressing the
fluid level below the level of oesophegeal sphincter.

NB:-Serotonin (5-hhydroxy tryptamine) is a product of tryptophan biosynthesis in the rumen has inhibitory effect on reflex opening of coudal oesophegeal sphincter.

*The gases mainly (coallaesence in free layer in the dorsal rumenal sac then and eructated through oesophegus by contraction of rumenal sac.
*So bloat resulted from either (prevention the collasence of gas in free layer → frothy tempany and /or prevent the eructation → free gas tempany.

<u>1-In G.L.B.:</u>

a)-Due to grazing of agricultural pastures containing clover or alfa alfa leads

to (Ruminal over distension/ obstruction of cardial oesophegeal sphincter by stable froth.

b)-Gas production occurs by:

*Acidification of dissolved bicarbonate.

*Decarboxylation of inorganic acids.

c)-Slime layer or surface active by:

-Dissolved leaf protein. -Salivary mucoprotein. -Protozoal protein.

d)-Slime layer leads to prevention the eruption of gases and cannot escape in free layer → over distention of rumen by frothy material.

2-In H.L.B.* Similar to green legumes bloat but it's due to feeding on whole chopped, ground or pelleted alfa alfa or clover hay which is especially conductive to bloat when highly leafy, recently cured combined with barely and constituting more than 10% of the ration.

3-In G.C.B.

1-Resulted from feeding on bloat producing concentrates especially corn, barely, soybean meal and ration containing less than 10% of roughage. Also favor development of bloat.

2-Gas produced from (aodifing of dissolved bicarnonate, microbial fermentation)

3-Slime layer formed by (Bacterial capsular slime composed of nucleo proteins and polysaccharides).

4- Deficiency of mucin in the saliva may leads to bloat (mucin act as anti foaming agent).

***N.B* *** GLB, H.L.B. and G.C.B. lead to hindrance the collection of gas in free layer du to the active surface slime layer so it leads to "forthy tempany" this forth leads to bloking of oesophegeal sphincter.

4- In Free Gas Tempany:

1-Occurs when there is hindrance in scaping of gas from the rumen as in case of:

-Ruminal atony or diseases cause it as fever.

-Pressure on the cardial sphincter of the oesophegus due to diseases of the diaphragm and lungs causing respiratory disorders and increase the interathoracic pressure

NB. -Increase the interarumenal and intera thoracic pressure leading to pushing of the blood to the prepheral circulation away from vescera helping in increase the dissolving Co_2 in the blood → Acidosis or academia.

- Acidity in the reumen about "6-5 PH" (N = 6,7Ph).

Clinical Signs:-

1-In frothy bloat the signs are developed rapidly within few hours after eating of bloat producing-food, so many cases escape clinical observation.

2-Pulgging of left paralumbar fossa due to increase the intera rumenal pressure.

3-Discomfort animal and may kick the abdomen.

4-Lying down and getting up again.

5-Within few minutes the condition is exacerboated and breathing becomes

difficult, shallow, rapid and oral.

6-Cyanosis of mucous membrane due to acidosis.

7-Ruminal movement by ausculation is depressed.

8-Morbidity 25% and mortality up to 50% of affected animals.

9-Neglected animal die from acidosis or respiratory distress

10-Free gas tempany may attack again.

PM Lesions:1-Over distended abdomen of dead animal.

2-Distension of the rumen with foam and viscous ingesta.

3-Thorax and abdomen contain blood but rumen contains echymotic haemorrhage due to rupture of some blood vessels due to over pressures.

4-Cyanosis of cervical part of oesophegus and blanched in thoracic part.

5-Rupture of rumen, diaphagm or abdominal wall may occur.

Diagnosis:-

1-History of eating of bload producing food, clinical signs and PM lesion.

2-By using of stomach tube to differentiate between free gas tempany and frethy tempany by:

*The pressure is completely reliefe by stomach tube only in free gas tempany.

*Hisotry of previous attack of free gas tempany.

3-By perccusion of abdomen → tympanic or drum sound.

*Auscuttation → ruminal stasis.

*Normal rumenal movement 3-6/2 min. in sheep and 2-4/2 min. in goat

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Treatment and Prevention:

1-In case of forthy tempany:

*Converted the forthy tempany into free gas tempany by using of anti-foaming agent as R1-Secodine R1 Bloatremidy R1 Gas remidy.

R1-Dimethicone R1 Simethicone. Or using of formalin or kerosen about 20ml to overcoming the surface tention as kerosen leads to decrease the surface tention and consequently help in eruption of gases and coallase in free layer.

2-***Using of trocar and conula*** to reliefe pressure or by surgical interference by reumenotomy.

3-***Mineral oil or vegetable oil*** 0.5-1 liter to evacuate the rumnal contents.

4-Sod bicarbonate 2.5 to 5% I.V to over come the acidosis.

5-In secondary tempany we should remove the cause and if it's impossible the animal should be sloguhtered to avoid the risk of continuous expense or fatal attack.

6-We should withdraw the causes of bloat (bloat producing food) and providing of balanced ration (quantity and quality).

7-Ruminal tonics as :-- R/Digastin - R/Vapcodigest - R/Vetadigeste.
-R/Hepovet - R/Stomatom - R1-Bekahepar.

8-Anti histaminic as :- -R/Tavegyl -R1 Avil -R/Vetibenzamin.

5- Ruminitis

Definition: Acute or chronic focal inflammation of the rumen caused by endogenous or exogenous irritant or microbail pathogens.

Occurrence:1-Ruminitis occurs in all breeds, sexes and ages, but more common in the feed lots lambs and kids 4 to 9 months of age than other age groups.

2-Usually during the early stage of fattening specially during summer and fall specially when lambs move into feed lots.

Etiology:- Ruminitis *Usually caused by* :-

A)-Fermented lactic acid. B)-Some poisonous as heavy metals

C)-Some viruses such as that cause contagious eczyma or poxiviridae

Pathogenesis: 1)-The disease usually begin with the action of lactic acid (that produced usually due to lactic acidosis) or virus or poisonous heavy metals on the mucosa.

2)-Concentrated irritants on mucasa leads to it's inflammation and ulceration and necosis of the ruminal wall.

3)-The continue movements of the rumenal contents leads to errosion of the ulcerated or necrotic mucosa and may reach the lamina propria and stimulate formation of granulation tissues.

4)-Bacteria of the rumen specially ubiquitous fusobacterium necrophorum can penetrate the wall of rumen and reach the blood circulation and may

localized in the liver leading to abscesses formation in the liver.

Clinical Signs: 1-Ruminitis of small or moderest extent may produce no signs. 2-Sever extensive lesions specially when associated with extra ruminal lesions as stomatitis or hepatic abscesses it leads to :-A)-Depression B)-Anorexia C)-Ruminal immobility (stasis) D)-Loss of body weight 3-Morbidity 10%. 4-Course of the disease about 10 days. 5-Mortality of untreated animal up to 20%.

Diagnosis: ·Difficult to be diagnosed clinically but can be detected ***by PM lesions*** as 1-Acute inflammation and chronic ulcers in the anterio ventral sac.
2-Acute or chronic or both lesions may present in one animal.
3-Acute form: (Hypermia, necrosis, odema, sloughing of papillae).
4-Chronic form (granulation tissue formation and scare formation and liver abscesses may be present).

Treatment and Prevention:

1-Graduale change of the ration of feed lots lambs from roughage into 90% concentrated ration.
2-Controlling and treatment of lactic acidosis to decrease the amount of irritancte lactic acid.
3-Vaccination against contagious eczyma or against other cusative viruses.
4-Oxytetracycline in ration (50mg/hea/day) to prevent ruminitis and liver abscesses.

6-Ruminal Para Keratosis

Definition:-

Nutritional degenerative change in the mucosa of rumen characterized grossly by browning hardening, thickening and clumping of the ruminal pappliae, and microscobically by accummulation of excessive layers of keratinized squemous epithelial cells on the papillary surface, and caused by diet of finely ground feed.

Occurrence:-

1-In all breeds and sexes of fattening animals specially3-9 months.

2-Usually more common in winter season because it's season of fatting.

Etiology-:

-Feeding on finely ground feed without pellets or with less than 10% roughage for long period may cause ruminal para keeatosis.

Pathogenesis:

1-Ration with increase of finely ground feed (concentrate) and decreased roughage leads to change in the numen as:

A)-Lowering of rumenal PH→ acidosis of rumen.

B)-Alteration of the proportion of the volatile fatty acids with increase the production of butyrate and lactate.

2-Lactate converted into lactic acid-and may leads to lactic acidosis.

3-Butyrate leads to disturbances in normal rumenal keratinization and causes rumenal parakenatosis.

4-The normal physical abrasisve action of roughage on the ruminal papillae play an important role in prevention of abnormal keratization.

5-The normal stratified, keratinized epithelium of the ruminal mucosa act as a protective barrier and play an important role in ruminal biological transport and metabolism ,so abnormal keratinization of rumen mucosa leads to defects on ruminal absorptive efficacy and metabolism.

Clinical Signs and PM Lesiones: -There are no speicific clinical signs:

***PM**:* Browing, hardening, thickening and clumping of rumenal papillae.

Histopathologically: show accummulation of excessive layers of keratinized squamous epithelial cells on the surface of the rumenal papillae.

Diagnosis:-

1-Difficult to be diagnosed clinically by veterinarian.

2-PM lesions and histo-pathological change.

3-Differential diagnosis with hypo vitamenosis-A.

Prevention:-

1-Avoid feeding on complete ration of finely ground feed without roughage at least 10% of the ration.

2-Menonsin and lasalocide (Ionophore antibiotices) give good result in prevention of rumenal parakeratosis as they decrease the production of lactate and butyrate while increase the production of propionate.

7- Abomasal and Doudenal Ulcers

Definition:-

It's focal disruptions on the mucous membrane of the abomasum and duodenum which usually as secondary complications to some acute disease as salmonellosis, blue tongue and lactic acidosis.

Occurrence:-

Affect all breeds, sexes and ages specially 3-6 months of feedlots lambs specially during late summer and fall.

Etiology:-

1-Causes of primary ulcers are unknown but we considered that from hypersecretion acting on area of mucosa with lowered resistence.

2-The factors that may contributed are:

(A)-Haemorrhage in the lamena propria of the stomach or intestine.

(B)-Penetration of nematod larvae

(C)-Some stress factors as sudden change in the diet from roughage to concentrates

(D)-Mucositis. *(E)-Defective covering mucus.*

Pathogenesis:-

1-The Etiological agents that mentioned before leading to dvitalization of the gastric mucosa so, the gastric juice leading to gradual digestion of the devitalized epithelial cells.

2-Just the covering epithelium is desteroid the gastric juice lead to

digestion of most deep layer (mesodermal cells).

3-In the advanced case the ulcers may reach the lamina propia and may penetrate the gastric wall or duodenal wall causing fatal periotenitis.

4-The errosion if it includes large blood vesele it leads to fatal internal haemoorahge.

Clinical Symptoms:-

1-Ulcers that not accompanied with haemorrhage the animals may show anorexia and signs of pain as kicking of abdomen.

2-Ulcers that associated by haemorrhage the animals show:

*Haematochezia(presence of blood in the faeces)

*Anaemia *Weakness.

3-Perfeorating ulcers lead to periotenitis with fever and abdominal distension from gas in the abdominal cavity.

PM lesions:

1-Ulcers in gastric or duodenal mucosa up to 20mm in diameter.

2-Perforated abomasal or duodemal wall in case of perforated ulcers.

3-Peritonitis and internal haemorrhage, may be present.

Diagnosis:-

Form PM lesions usually.

Prevention and Treatment:-

Have not been developed.

8-Lactic Acidosis or Impaction

Synenoms:

1-Grain engorgment 2-Ruminal acidosis 3-Acute indigestion

4-Founder = laminits. 5-Grain overload.

Definition:-

*It's acute metabolic disease of sheep and goats characterized clinically by → inappetance-depression, lameness and coma.

*Biochemically by → Acidosis,rumenitis, acedemia, and haemocentertion. Caused by sudden engorgement of grains or other easily fermentable substances by the animal not accustomed to such food in large amount.

Occurrence:-

1-In all breeds, sexes and ages of sheep and goat .

2-More common in feed lot lambs and kids 3-9 months than other age groups.

3-More common in the farm flock farms than yard or pastured systems.

4-More common after storms when the animals have been unable to eat for periods of time followed by feeding in unlimitted quantities of high concentrates feeds.

Etiology and predisposing factors:-

1-Lactic acidosis is caused by engorgement on the following feed By the animals that not accustomed to such food in large amounts:

-Highly fermentible carbohydrate rich diet,as barely, wheat, or corn

-Sugar beets, sugar cane.   -Mangoes, potatoes…..etc.

2-Sudden change in the diet from 100% roughage to 90% concentrates.

3-Miscalculating of ration constituents specially the concentrates parte.

4-Feeding on large amount of concentrates after period of anorexia, thirst or after accidental fast.

5-Inadequate distrebution of concentrates in the pens, so the animal separates the concenterates from roughage specially in poorly mixed ration.

6-Change in the ration ingredient i.e. high in grains and low roughage (normally roughage not less than 10%).

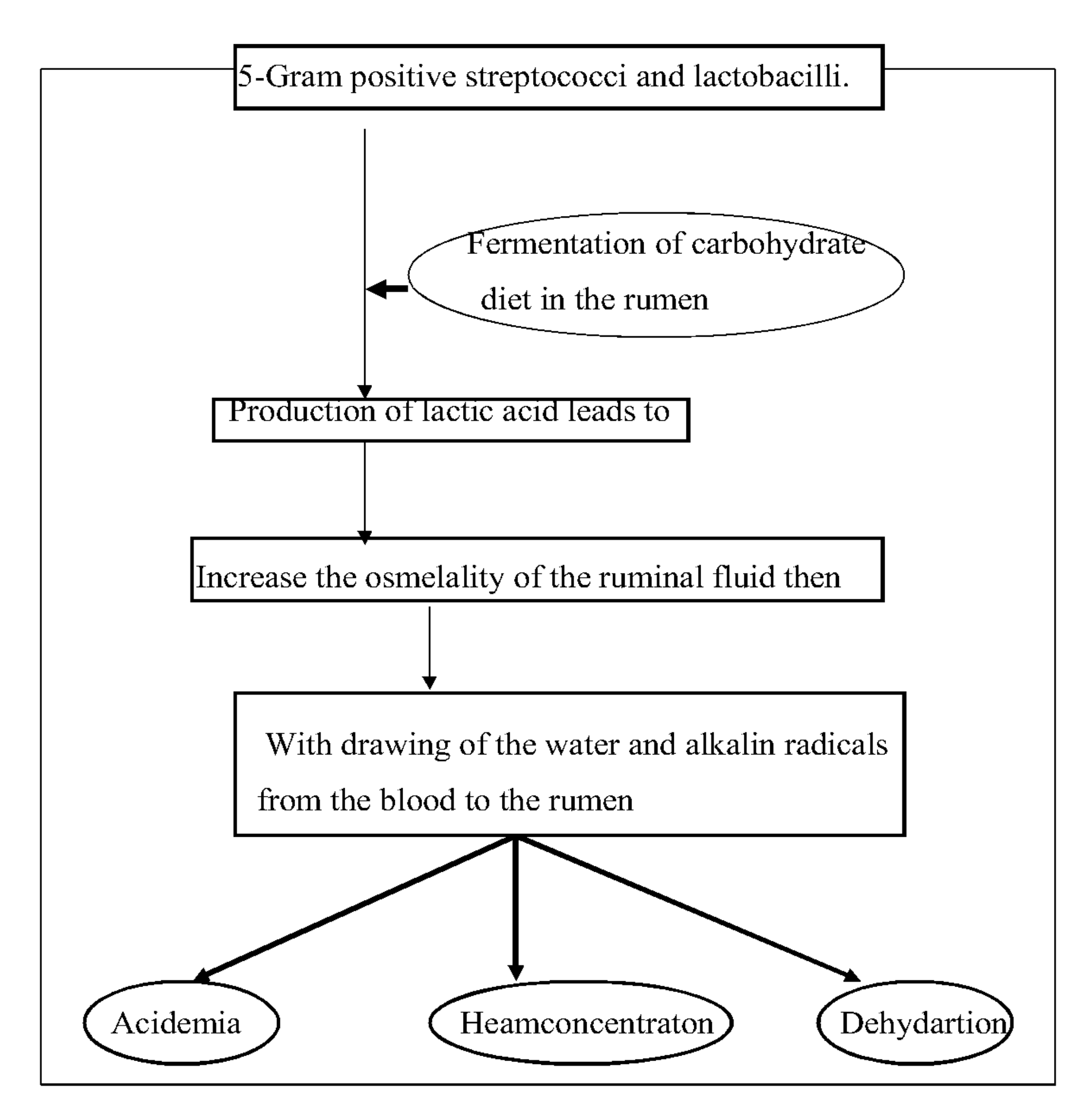
Pathogenesis: - (Also see part (2) large rumeninat medicine)

1-Pathogenesis of lactic acidosis is a series of biochemical changes in the rumen and blood.

2-Excessive microbial produciton in the rumen and subsequent, absorption of lactic acid is responsible for lactic acid syndrom.

3-Normally the lactate is low in blood but it's increased as a result of excessive production of lactic acid from formentable feed in the rumen.

4-When sheep or goat fed on hay or grass [cellulose food] the cellulytic microorganisme (Gram Negative streptococci) act on such food. But when following by engorgment of starch-rich food , so the amylolytic grame positive strepococci and lactobacilli rapidly multiply and then replace the gram negative bacill.



6-Haemconcentration and dehydration of the tissue enhance the spleen to evacuate large amount of it's RBCs storage.(Increasing of P.C.V up to 55-60%).

7-Excessive production of lactic acid leads in the rumen with increase the contentes of the ruminal fluids lead to:-:

(A)-Ruminitis, ruminal ulcers or ruminal parakeratosis para keratosis. (please reveiw this diseases again)

(B)-Perforation of ruminal wall leading to peritonitis or liver abscesse.

(C)-Absorbed through the rumenal wall to the blood leading to systemic acidosis or academia.

(D)-Secreted in the urine leading to acidfied urine

(E)-Acidosis leads to histamine release causing lamenitis or it may leads to polioencephalomalica (nervous signs).

(F)-Lowering the ruminal PH leading to: -Death of microflora.

-Decrease the ruminal movement when PH. 5 or PH.3-4 It leads to complete ruminal stasis. -Prevent production of fatty acids (milk fat) → decrease the milk production.

(G)-Increase the size of rumen leading to:

-Pushing the blood to the peripheral circulation causing high solulability of Co_2 → acidosis or Acidemia.

-Press on the diaphragm and lungs causing respiratory distress.

8-Death usually due to haemconcentration, acidemia or dehydration.

Clinical Symptoms:- 1-Diseased animals separated from the flock.

2-Depression, dullness, and weakness with incoordination in the movement. 3-In appetance and may anorexia.

4-Body temperature normal or may slightly elevated

5-Accelerated pulse and respiration, due to systemic acidosis.

6-Ruminal stasis with skin dehydration.

7-Lameness and the animal reclined then coma and death (the dead animal usually present in lateral recumbency).

8- Sunked eyes with lowering of the head due to dehydration.

9-By palpation → doughy material in the left flank region.

10-Abdominal distention specially in left sub lumber fossa.

11-By auscultation → complete ruminal stasis with some gurgling sound.

12-In some advanced state diarrhoea may occur (good prognosis).

13-In late stage there are paddling and convulsions → death

14-Fluid splashing sound may be present by auscultation of the rumen.

Diagnosis:- 1-History and clinical symptoms.

2-Palpation, auscultation of the rumen and skin fokl test (to detecte degree of dehydration).

3-Laboratory diagnosis: ****Ruminal juice analysis:***

-Acidic 3.8-6 (N = 6.2-7.2) -Disappearance of microflora (by microscopical examination).

-Increase lactate contentes and plasma fluid.

-Darke blackish coloured ruminal juice.

-Presence of Gram positive cocci and absence of Grame negative one

****Blood analysis:*** -Increased P.C.V. up to 60% (N = 45%).

-Low PH. (N = 7-7.2). -High lactate contents.

****Urine* analysis:-**-Oliguria -Proteinuria -Acidic urine low PH.5 (N=8)

Treatment:- *- The main pointes of the treatment are :-*

(A)-Hygienic treatment. (B)-Evacuation of ruminal contents.

(C)-Correction of acidosis. (D)-Correction of dehydration.

(E)-Ruminal tonic(for improving the contractility of the rumen)

(F)-Antihistaminic for competing the lameness

1-In early stage → magnesium hydroxid and penicillin orally for removing the ruuminal contents and killing of gram positive microorganisms.

2-Liquid parafin up to ½ liter then give mag. Sulphat 1g/1kg Bwt.

3-Correction of PH. By: (A)-Sod. Bicarbanate 2% 200ml I.V.

(B)-Magnesium hydroxide or mag. Bicarbonate 150gm orally.

4-Correction of dehydration by slain sol. 1-2 liter I.V.

5-Anti-histamenic [avil, Allecure, Vetebenzamine, or Tavygel].

6-Ruminal tonices [Stomatan, Vapcodigest, Vetadigest…..etc]. carbamyle cholin chloride or neostagmen or physostagmen (para-sympathomimetic) to improve the ruminal contractility.

7-Avoid Ringer because it's increase the acidity, because it converted into lactate which converted into lactic acid and increase the acidity.

Control:- -Competing or avoiding and correction of the etiological and predisposing factors.

N.B. Mag. Hydroxide or sulpate considered as saline purgative as it leads to increase the water contents of the intestinal lumen and consequently heads to increase the prestalitic movement of intestine and evacuation of G.I.T. contents. (I did not prefer its use from my point of view).

9-Enteritis

Definition: Enteritis means inflammation of the intestinal mucosa resulting in malabsorption, diarrhoea, dehydration and acid base in blance.

Etiology:- Summerized in the following table:-

(A) Bacterial	***Incidence***	***Symptoms***
1- E-Coli 2-Clostiridium preferengin type B- lambdysentry)	-In newly born lambs - Not more than 10 days of age or up to 2ws.	- Acute yellow diarrhoea - Toxemia and death
2- Salmonellosis	-Young lambs -In adult specially in late pregnancy	- acute diarrhoea. - Toxemia, fever & death
(B)Viral 1- Rota and corona virus	- Young lams .in over crowded area. Lambs 10ws of age.	-Acute diarrhoea white -Septicemia-abortion. - Acute diarrhoea.
(C) Parasitic:		Diarrhoea,weight loss
1- Ostertagia spp. 2-Trichostrongylus 3-Fascioliasis	- Older ewe. - All ages. -After 4-9 months of age After weaning or beginning of fattening	-Inflammation,of abomasum. -Chronic diarrhoea. -Icteric mucous.
D) Protozoa: 1- Coccidiosis 2- Gryptosporidosis	-In bad hygiene & overcrowding flock -lack of colostrum	Haemorrhagicdiarrhoea -Dystentry-death. -Death within 2-3d. -- -Anorexia.

Pathogensis:-

(I)-The intestinal tract receive the nutrient, chloride, electrolytes and other fluid which come from the saliva, stomach, pancreases, fluid intake and from the intestinal tract itself, so any dysfunction of the intestine as inflammation leads to the following:

(A)-Change In The Osmolality:

*Accumulation of the fluid in the intestinal lumen leads to increase the asmotic pressure causing withdrawing of the water contents from the body tissues and increase the water in the intestine accompanied with inflammation of the intestinal mucosa so diarrhoea occurred with dehydration.

B- Change InThe Absorption:

*Inflammoation of the intestinal mucosa leads to destruction of the intestinal vilia and consequently reduction of the absorption.

C- Increase The Perestaletic Movement:

*As a response to inflammation and to getride of excessive amount of water that present causing diarrhoea.

(II)-In case of acute diarrhoea lose of body fluids leads to:

*Haemoconcentration which can be detected by increased P.C.V.

*Decreased the amount of circulating blood lead to peripheral circulatory failure due to increase of blood viscosity.

*Decrease the renal blood flow which manifested in olig-or anuria as compensatory reaction of loss of the body fluids, and increase of blood urea nitrogen (BUN).

1- The body tries to compansete the circulatory failure by increasing of protein, fat and carbohydrate catabolism resulting in produce from of large amount of wast products such as lactate which converted into lactic acid causing acidosis or acidemia.

2- Decrease in the electolytes as sodium, potassium, bicorbonate… …..etc. causing acid-base imbalance.

3- Decrease in the protein causing hypoproteinaemia.

(III)- Enteritis may considered as secondary causing of other may disease as hypocalcemia, hypo phosphatemis, hypocuprosis…………etc.

Clinical Findings:-

1-Diarrhoea or dysentry – Enophthalmia i.e. reduction and retration of the eye ball.

2-Fever if the caused infectious.

3-Anorexia, wearkness and death may occur.

4-Dehydration which manifested in form of:-

-Sunken eye.

-Shrunken of skin.

-Loss of skin elasticity.

-Increase the capillary refill time.

5-Temperature according to etiology.

6-Other signs of specific etiology.

N.B. Management errors may leads to diarrhoea as in cas of :

*Sudden change in diet.

*Feeding on young darawa.

*Drinking of so hot or cold water.

Diagnosis:-

1-Skin fold test and capilary refil time test):-

2-Case history and clinical findings.

*3-**Laboratory Diagnosis:-***

a-Blood picture [Anaemia-haem concentration – P.C.V]

b-Isolation of the virus or bacteria.

c-Fecal analysis.

d-Biochemical analysis for sodium, postassium, protein…….etc.

Treatment:-

"According to the etiological factors"

1-Parasetic → Anthelmentics.

-Protozoal → sulpha drugs

-Bacterial → anti-microbial drugs.

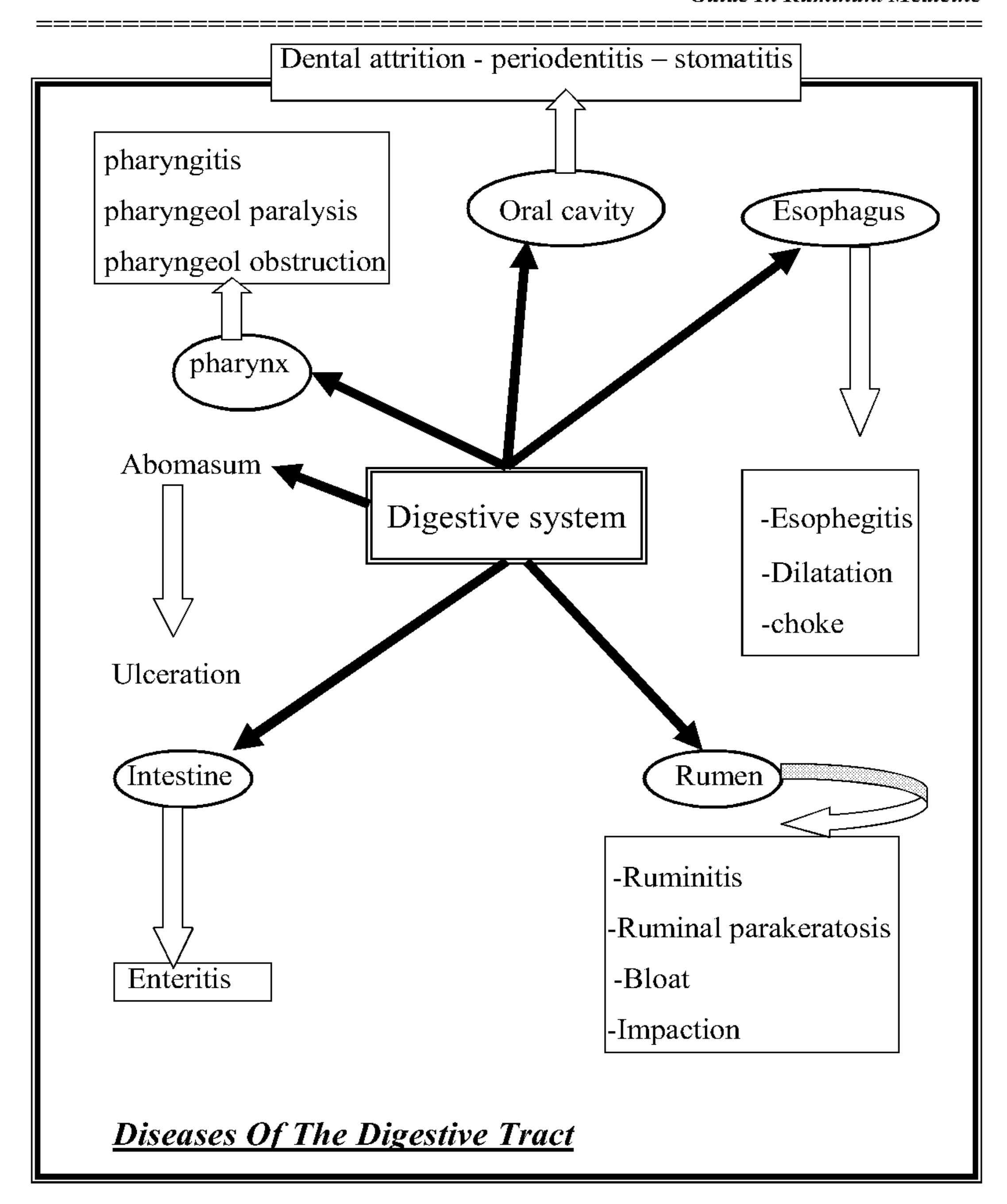
2-Koalin and pectine mixture as intestinal coater and sedative.

3-Treatment of dehydration by fluid therapy.

- Isotonic nor male soline solution 0.9% I.V.
- Dextrose 5% I.V
- Isotonic sodium bicarbonate in water 1-3% I.V.

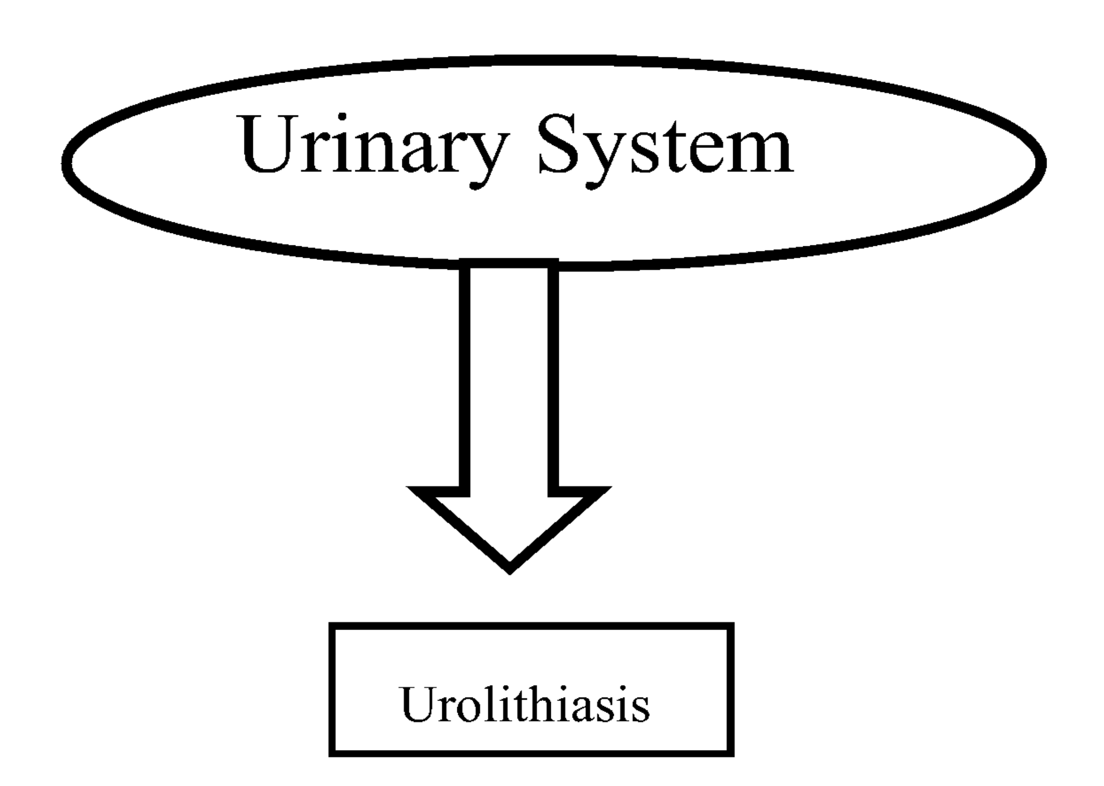
4-Withdrawing of food at least 12 hours.

NB. Also see part (IV) dehydration



Diseases Of The Digestive Tract

Chapter No.4



Urinary Calculi or Urollithiasis

Definition:- It'one of the most important disease that effecte the urinary system in the small ruminanat (males common) characterized by:

1-Formation of struvitic concentration composed of matrix and minerals within the urinary tract.

2-Retention of urine 3-Occlusion of urethera.

4-Rupture of urinary bladder or urthera.

*Caused by imbalance of dietary minerals and other factors as hormonal & environmental factors.

Occurrence:- 1-Occurs in all breeds and sexes of sheep but more common in 3-6 months of age of feedlots lambs and kids (specially casterated animals).

2-Most cases develop during autumn and winter particularly following a periods of inclement weather. In all sheep and goat producing countries.

Etiology And Predisposing Factors:-

*Calculosis usually due to an inter action of dietary, hormonal and environmental factors.

1-Feedlots economic requires to fattening the animal and marketing in minimum time ,so the ration contains 70-90% concentrates.

2-The grains specially wheat and sorghum contains of high percentage of phosphorous and magnesium.

3-Urinary tract infections may contribute to the production of uroliths by

increasing the urine 'PH' ,so providing environment in which phosphate and magnesium ions less soluble and ammonium ions are more valuable.

4-Animals on high grain diet with minerals imbalance (high phosphorous and low calcium) leads to formation of phosphate (struvite) calculi containing calcium, magnesium and ammonium phosphates.

5-Water deprivation may contributed to calculi formation due to it leads to high concentrated urine.

6-In arrid area where the general hay or grass are the main dietary components ,so silicate (SO_2, H_2O) calculi are commenly formed.

7-Cold weather predispose in calculi formation due to contraction of penis & urethria in the cold weather.

8-Feeding on subterranean clover which is high in calcium leads to formation of calcite calculi ($CaCo_3$) i.e. calcium carbonate.

9-Diet that rich in oxalate leads to formation of oxalate or calcite calculi.

10-Casterated rame highly susceptable to affected with calculosis due to:

a-The diameter of urethera in casterated rame is narrow than that of non casterated one .

b-Osterogen level increase in casterated animal that leading to metaplasia of uretheral epithelium and enlargement of accessory genital gtands that also leads to narrowing of the diameter of the urethera.

c-Testesteron in non-casterated animal help in widding the diameter of urethera.

11-Females less susceptible due to wide and short urethera ,so any calculi

is pushed out with micturation.

Pathogenesis:- Pathogenesis of urolithiasis is a complex action between: Dietary – Homoral – Enviromental factors.

(I)-Dietary Factors:

1-Feeding on concentrates for long period may predisposing of urolithiasis as it contains high percentage of phosphrous than calcium and the ratio between calcium and phosphorus is near.

2-Feeding on plant which grow on dried area it's usually rich in silicate which leads to increase the level of silicic acid in the urine causing formation of silicate calculi.

3-Feeding on subterranean clover which high in calcium carbonat which enhance formation calcium carbonate calculi

(II)--Homonal Factors:

1-Urolithiasis is more common in casteated rame than non casterated one that because of testesteone hormon leads to dilation of the urethera and consequently decrease the incidence of urolithiasis.

2-Esterogen level was increased in casterated animal that leading to hyperplasia of the urethera and epithelium and enlargment of the male accessory genital gland that causing narrowing of urethera.

3-Urethera in male is long and narrow than that of female which is short and wide.

4-Presence of sigmoid flexure and uretheral process in male considered as predilection sites of urolithiais.

(III)-- Enivronmental Factors:

1-Exposure to cold weather for long period leads to spasm of the urethera and narrowing of the diameter.

2-Long period of lacking of water leads to increase the concentration of urine and precipitation of salts.

3-Bacterial infection of the urinary taract leads to increase the urine PH which enhance the precipitation salt as phosphorus.

Types of Calculi:-

1-Stuvite magnesium and ammonium phosphate.

2-Calcium carbonate (calcite). 3-Oxalate.

4-Silicate (Sio_2-H_2O) 5-Amorphous phosphate.

Pathogenesis(Formation of the calculi)

a) Nidus formation (Nuclous of calculi) or Matrix.

b) Preciptation of solutes.

A- Nidus Formation:

1-Usually formed from group of desquamated epithelial cells that due to either :- a-Infection of the urinary tract.

b-Vitamin-A deficiency (appear in large numbers of animals)

c-Administration of estorgen that leading to excessive desquamation of epithelium of the urinary tracte.

d-When stilbesterol used as growth promoter.

2-It's usually organic matter (mucoprotein, specially mucopolysaccharide).

B- Preciptation Of Solutes:

1-High PH-urine (Alkaline urine) help in periciptation of phosphate leading to formation of phosphate calculi.

2-Increase the concentration of silica in the diet leads to increase it's concentration in the kidney leading to the urine of this animal becomes supersaturated with silicic acid causing polymerization or pericipitation of silicic acid and calculi formation (Silicate formation).

3-Depriviation of the water intake leads to increase the concentration of the urine and promote the pericipitation of solutes.

(IV)-Urolithiasis may occurs in kidney ureters, urethera or urinary bladder, and complete obstruction is fatal because it leads to rupture of urinary bladder specially when the calculi is spherical large in size leads to complete obstruction of urinary bladder (within 2-3 day of untreated animal).

(V)-Rupture of urethera is more common specially when the calculi is irregular in shape leads to partial obstruction with pressure necrosis on the wall of the urethera.

(VI)-Ureamia may occur and leads to nervious manifestations due to high level of ammonia which responsible for neurologic symptoms which associated with urolithiasis.

Clinical Signs:-

1-Straining during urination or dysuria (painful urination) or stranguria (slow painful nurination) specially in incomplete obstruction.

2-Drippleing of urine and the urine is tainged with blood.

3-In case of complete obstruction the prepuce hair is dry and fully distended bladder can be palpated through the abdomen.

4-Uretheral pulsation at ischial arch and can be detected rectally by digital palpation.

5-Movement of the tail form side to side with kicking of abdomen.

6-Groning sound and laying down and getting up again frequentaly .

7-Anorexia and isolation form the flock

8-Neglected untreated case ended by rupture of bladder or urethera according to location of calculi and it is usually accompanied by relief of the signs of pain.

9-All mentioned symptoms are disappeared and the animal become relaxed after rupture of urinary bladder or urethera.

10-Ruptured urinary bladder leading to abdominal distension with urine *"water belly"* and that urine can be detected by exploratory puncture of abdomen and the adour of urine can be detected easy specialy after heateing of the aspirated fluid from the exploratory puncutre.

11-Ureamia may occurs that characterized by urinefrous odor of breath.

12-In advanced case shallow rapide respiraton with rapide pulse rate may be occurs.

13-Anurea in case of complete obstruction.

14-Rectal prolapse may occurs due to straining during urrination .

PM lesions: 1-Abdominal cavity contains large amount of urine.

2-Ruptured urinary bladder or urethera.

3-By dissecting the urethera (specially segmoide flexure or Uretheral process). In the urethera the site of obstruction characterized by necrosis with haemorrhage.

4-Beginning of hydronephrosis may be evident.

Diagnosis:- 1-Case history, clinical symptoms and P.M lesions

2-Laboratory Diagnosis:

(A)-Blood (blood urea nitrogen, plasma protein, potassium, creatinine, phosphate) increased.

* Sodium and chloride level are depressed.

(B)-Urine: (presence of RBCs & epithelial cells)

(C)-High level of creatinine and B.U.N. from aspirated fluid from abdomen by paracentesis.

Prevention:- 1-Providing rations contain 2:1 calcium to phosphorus and calcium chloride should be added to the diet to adjust the calcium : phosphorous ratio.

2-Addition of common salt (sodium chlorid) to the ration 1 to 4% up to 9% can be added as Nacl prevent the formation of silicates calculi by reducing the concentration of silicic acid in the urine and maintaining it below the saturation concentration of the solutes in the urine.

3-Addition of ammonium chloride [2% of diet and 10gm/day] that prevent the formation of phosphates calculi by increasing the acidity of urine so leads to solubility of phosphate and magnesium

(soluable in acidic urine), also ammonium chloride prevent the formation of silicates calculi. 4-Supplementation of adequate amount of fresh water. 5-A well balanced diet include adequate amount of vitamin-A.

Treatment:- ***1-Anti-spamodic as:***

*R/Buscopan, Spacomcine, Glucalynamin. Ampl injection IM or IV.

*R/Spasmo paralgine, Analgine, Novalgins.

*R/Spasmo canulase….etc or Atropin sulfat (0.05-0.5 mg / Kg.BWt. i.e. 1 ml. / 20-200 Kg.Bwt. in the concentration of 1%) S/C. or IM.

2-Acidification of urine :- by ammonium chlorid 7-10mg/head/day of lamb 30 kg Bwt. to dessolve phosphate calculi.

3-Fluid therapy :-as saline solution or dextrose I.V.

4-Surgical interference by:*Amputaion of uretheral process (common predilection site of formation of the calculi).

*Perineal uretherotomy proximal to the site t of obstruction.

*Partial cystotomy. N.B. Type of calculi according to urine "PH":

A- Acidic urine	***B Alkaline urine***
1- Amorphous urate (Unreconzed shape) 2- uric acid ("Whetstone"shap) 3- ammonium urate (Thomy apple shap)	1- Triple phosphate. (Prysmetic shap) 2- Calcium oxalate (Envelope shap) 3- Calcium carbonate (Dumbel shap)

Chapter No.5

Respiratory System

1-Rhinitis

2-Nasanl Myasis

3-Pulmonary adenomatosis

4-Pneumonia

5-Ovine adenomatosis

6-Mycoplasma Pneumonia

7-Clamydia pneumonia

1-Rhinitis

Definition: Rhinitis means inflammation of nasal mucous membrane characterized by sneezing, wheezing and abnormal sound during inspiration as well as nasal discharge which may be serous, mucoid or mucopeurelant according to etiology.

Etiology:

1-Oestrus ovis (nasal bot) 2-Sheep pox 3-Contagious ecthyma (rare)
4-Allergic rhinitis. 5-Blue tongue 6-Melodiosis
*Atopic rhinits (means that defects in immuno system causing allergy as rhinits).

Pathogenesis:

1-Rhinitis is of minor importance except when it leads to obstruction of the nasal cavity leads to difficult in respiration.
2-Inflammed nasal mucous membrane may be erroded and may help in secondary bacterial invasion.
3-Rhinitie itself not considered as a disease but it may considered as a complication of other diseases as pox.contagiuos eczyma …………etc.

Clinical Symptoms:

1-The cardinal (main) signe of rhinitis is nasal discharge which is serous firstly and become mucoied and after bacterial invasion it become peurulent.

2-Erythema, errosion or ulceration may be observed by inspection of the nasal cavity.

3-Sneezing followed by snorting sound.

4-The disease may unilateral or bilateral.

5-Mouth breath in case of bilateral affection.

6-The animal may rub it's nose along the ground due to irritation.

7-Symptoms of specific disease as pox, nasal myiosis……etc.

Diagnosis: 1-Clinical signs and case history.

2-By using of flexible fibrooptic endoscope or rigid endoscope.

3-Observation of larvae in case of oestrus ovis infection.

4-Differential diagnosis from inflammation of the facial sinuses in which the nasal discharge is continious and persist and there is no signs of nasal irritation.

Treatment:- 1-Treatment the specific disease.

2-Removing the tanacious excudate which causing obstruction of the nasal cavity and flushing the nasal cavity by saline solution or worm water.

3-Nasal decongestant sprayed up into the nostrils may provide some relief

R/vibroseal R/prosoline.

4-Anti-histaminic preparations specially in allergic rhinitis.

R/ Avil R/Allecure R/ Tavegyl R/Vegebenzamine.

5-Systemic antibiotics to prevent the secondary bacterial invasion.

2-Nasal Myiosis

Synonomes:- 1-Nasal Bots 2-Head Bots 3- Head Grub

Definition:- It's chronic rhinitis and sinusitis of sheep characterized by persistent anoyance and mucopeurulent discharge caused by oestrus ovis fly.

Etiology and Pathogenesis:- 1-Mainly oestrus ovis fly (the insect and larvae are both attack the animal).

2- Adult fly lives for 2-28 days in protected area such as property fences and building walls during that time each female mates and produce up to 500 larvae which on worm middays, she deposits on the nostrils of sheep.

3-The larvae enter the nasal cavity and feed on nucous and desquamated cells of the nasal cavity or may enter the frontal or maxillary sinuses. Then after 2-10 months the larvae return to the nostrils and sneezed to the exterio (in the soil it take about 26-36 days to be adult fly and then repete it's life cycle).

4-Attacking of fly and larvae causing persistent anoyance of the animals, so it research of cover itselef and reluctante to food.

5-Migration of larvae through the nostrils and sinsuses cousing irritation of the animals and may predispose of snusitis.

6-Presence of dead larvae may cause allergic inflammation of the nasal cavity or of the sinuses.

7-Some larvae may migrate to the trachea and lung and there produce

fatal reaction (rare).

Clinical Symptoms:- 1-Presence of the signs of rhinitis.

2-The animal shake it's head and rupping it's nose against inanimate object or on the ground or to other sheep, with chronic nasal discharge.

3-Isolation from the flock and off food (frenzied behaviour of sheep).

4-The animal seek cool areas such as damp shad where the flies do not frequent.

5-Morbidity → 80%, mortality → nil.

6-The course of adult fly attack periodically extends through the summer and the rhinitis and sinusitis continue up to 10 months.

7-Life cycle of the fly about 1-2months.

Diagnosis:- 1-History and clinical signs

2-Presence of larvae on the nostriles and sinuses.

P.M Lesiones:- odematous nasal mucous membrane & presence of the larvae in the sinuses.

Treatment:- 1-Sheep should be treated for nasal myiosis during autumn when most larvae are small.

2-Using of R1-Rafoxanide 7.5mg/kg Bwt. S/c (i.e 1ml/25kg. S/c)

R1-Invermectin (Ivomec-Iveen-Avemic) 1ml/25kg s/c

3-Antihistamenic (Avil, Allcure-Tavagyle).

4- Antibiotic to prevent the secondary bacterial infection.

4-Pulmonary Adenomatosis or pulmonary Carcinoma

Definition:- It's contagious neoplastic disease of sheep characterized by insideious but contineous development of weakness and emaciation, dypnoea, nasal discharge and fequent cough caused by retrovirus.

Occurrence:- 1-Occurs in all sheep-producing countries, all spp. And breeds especially marine sheep are susceptible and all ages and sexes.

2-The clinical signs appeared only in adult animals not in young one. (Neoplastic disease)

3-Goat also are susceptible.

Etiology:-1-Retro virus of the family retrovirdae (6 classes of the virus are isolated which are A, B, C, D, E and F).

2-The disease is transmitted by transplantation of all infected cell which coughed by diseased animal during coughing or blowing in the environment then in hale by healthy animal or the infection occur by taking the (v) itself.

3-Crowding sheep in pens specially closed buildings facilitates spread of infection from diseased to healthy animals.

Pathogenesis:- 1-The target cells of the (V) are:

-Type II alveolar epithelial cells. -Non-cliated bronchiolar epith. Cells. (rare)

2-In early stage the transformed asparted cells from diseased animal are proliferated forming a single layer lining the alveolar lumen then develop into papiliform mass that leading to narrowing the alveolar lumen → Hypox-dyspnoed-cough" normal.

3-The papilliform mass are proliferated further until fill the alveolar lumen then transformed from alveolus to other one.

4-The peripheral cells of the neoplasm contains secretory granules resembling to the normal sheep granular penumocyte, so these tumour cells ganulas secrete osmophillic secretion leading to formation surfactant production which when increase lead to increase the lunge excudate → nasal discharge so the increase the numbers of granular cells the increase the surfactant production.

5-The tumour cells undergo proteins synthesis and cells division with virus replication, and the small foci by expansion or coallesence together leads to forming of large nodules which may include the most of lung and the severity depend upon lung incapacitation.

6-After several months or even years metastasis may occurs to include the regional lymph nodes but the extra theracic organs are rare to be affected.

7-The growth of extensive neoplastic tissue within the lung may increase the resistance (hindrance) to pulmonary circulation that reflected on the right ventricle causing ventericular hypertrophy, failure and death.

8-During the long course complement fixing and neutrilizing antibodies are formed but not influence the progression of the disease.

9-The most immune cells that present in the disease are alveolar macrophage which play role in:

*Decrease the amount of surfactant material, so decrease the lung exudate.

*Limitting the metastasis of neoplasm.

10-Plasma cells which present in the interstitial space produced local immunoglobuline against sheep admomatosis.

11-Death occur due to:

*Sever hypoxia and dyspnoea.

*Ventricular hypertrophy or failure "CHF"

*Secondary bacterial invasion which may leads to death of premature animals.

Clinical Symptoms:

1-Depend upon the lung incapacitation.

(I)-In Early Stage:

-The animal in good physical condition.

-Shallow rapid respiration specially during work or exercise, with frequent cough.

-Extension of head and neck with each respiration.

-Dilatation of nostriles due to dyspnoea.

2-By auscultation → there is rales and lung consolidation specially in the venteral planes.

(II)-In Late Stage:

-Weakness, emaciation. -Nasal discharge.

-The animal remain in standing position because recumbence exacerbate the dyspnoea → death.

3-Incubation period → 2 months – 2 years, mortality up to 100%.

4-Course → several weeks to sevel months, morbidity up to 20%.

PM lesions:

1-In early stage → lung contain gray blue nodules 1-20 mm in diameter.

2-In late stage:

*Lung contain small nodules with large neoplastic mass.

*Regional lymphnodes are enlarged from metastasis.

*Right ventricle is hypertophied and dilated.

*Small amount of excavate may accumulated in some body cavities.

Histopathlogical lesions:

1-Proliferated alveolar and/or branchiolar epithelium.

2-Papillary mass are found on cuboidal epithelim

3-Affected bronchioles show both hyperplastic and neoplastic epithelium which also form papillary masses enterring the adjacent alveolus.

Diagnosis:

1-Symptoms, PM and hsitopathologic lesions.

2-Differential diagnosis: parasitic pneumonia, chronic progressive pneumonia and pastuerelosis all of these disease not produce nasal discharge but each has characteristic historpathological lesion.

Prevention:*Eradication and sloughtering of diseased and contact animal.

5-Pneumonia

Synonoms:- -Enzootic pneumonia -Pasteurelosis

-Heamorrhagic septicomia -Shiping fever

*Pneumonia usually multifactorial disease due to stress factors as:

A-Physical Stress:

*Bad limatic conditions. *Long transportation

*Fasting or starvation. *Sudden change of diet.

*Over crowding or herding and flocking

B- Bilogical Stress as:- *Vaccination. *Viral infection.

Definition:- *It's acute infectious disease of sheep affecting all ages with specially high prevalence in nursing and feedlots lambs, characterized clinically by respiratory distress, mucopurlent and may be blood tainged oculonasal discharge, depression, anorxia and pyrexia and pathologically by pneumonitis and pleuritis (incase of pleuropneumoria).

*Pneumonia is caused by complex interaction of environmental factors producing stress and a variety of micro-organisms working synergistically to damage the cell lining the respiratory tract allowing colonization and invasion of other organisms and a compensated host response.

Occurrence:(I)-Pneumonia commonly occurs in all breeds, ages, and sexes of sheep = goat throughout the world.

(II)-The prevalence varies from region to region and is influenced by the environmental factors and management practices.

(III)-Although all age groups are susceptible but the prevalence is high among nursing, weaned and feedlots lambs and kids.

*Presence of the sheep and goat in crowded area has some common risk factors as:

1-Easily transmission of the disease from diseased to healthy animal.

2-Build-up of potential pathogene in environment.

3-Production of harmful gases as Co_2, H_2S and amonia which irritate or damage the respiratory mucous or delay or alter the phagocytic properties of macrophage.

(IV)-Seasonal variation of pneumonia depend upon enterprise, climatic conditions and mangmental procedures.

(V)-The peak of incidence of pneumonia in feed lot lambs occurs 1-3 ws. After arrival in the feedlot after being predisposed by stress of either.

*Weaning *Transportation *change in diet and coming with lambs of different origins.

Transmission:- 1-Some animals chlamydiosis in the upper digestive tract and excrete it within the faeces and other animals carry pasteurellosis in the upper respiratory tract and excrete it within the nasal discharge.

2-During crowding the microorganism contaminate equipment, feed, water and air and probably transmitted to healthy sheep by inhalation into the respiratory system or by ingestion into digestive tract

Etiology And Predisposing Factors:

1-The causes of pneumonia is a complex of chlamydia and pasteurella

haemolytica and/or multocida depend upon some stress factors which may be:

(I)-External stress factors as:

-Transportation. – Overcrowding -Vehical vibration

– Noise -Sudden change in the weather.

(II)-Internal stress factors as:

-Weaning -Fear -Fatigue. -Vacation and hungry.

2-In acute out break the pasteurella haemolytic is most frequent pathogene that isolated from diseased lung tissue.

3-It's virtually impossible that the massive numbers of pasteurella hemolytica to infect the normal animal due to the clearing action of the normal respiratory system therefore the disease process depends on a combination of event As: the viruses of respiratory system damage the lining epithelium by causing inflammation, odema and necrosis thus destroying ciliary action and mucous protection allowing bacteria and mycoplasma to invade the respiratory epithelium then multiply and causing the disease.

4-Most common respiratory viruses are:

-Parainfleunza type-3. - Respiratory syncetial virus.

-Adenoviruses & Reoviruses.

5-Other causes of pneumonia:

-Salmonella abortus ovis -Streptococcus zooepidemicus.

-Corynebacterium pseudo tuberculosis

-Verminous pneumonia dictycacaulus filare.

-Meliodiosis (pseudomonas pseudomallei).

Pathogenesis:

(I)-Pathogenesis of pneumonia is a complex interaction between stress factors and biological factors (viral and bacterial infection) as the stress factors leads to increase the level of cortisol which has immune-suppression effect which facilitate the entrance of bacteria as mycop lasma, pasteuella and chalmydia.

(II)-Pneumonia has 4 stages.

Stage of congestion:

1-Pneumonia usually initiate from the most lower ventral lobe by congestion and odema in the lung tissue.

2-After exposure to stress factors either external or internal or due to viral infection or both the chlamydia become virulent and penetrate the bronchial then multiply in the septa and epithelial cells and then initiate the pneumonitis by congestion and odema of lung tissue.

Stage of red hepatization:

1-In these stage leucocytic infiltration occurs (give appearance of the liver to the lung) the resulting dead macrophages and secretion of fibrinolytic enzyme. Which leads to lysis of the fibrous exudate and beginning of stage of resolution until the alveoli returned to it's normal form.

2-If there is no-leuckocytic infiltration due to bad immune status of the animal and untreated animal the pneumonia ended by consolidation of the lung or if it treated to leads to resolution (4th stage).

(III)-Extension of the lesion may occurs to the pleura or pericardium causing pleuritis or pencarditis as a complication of pneumonia → pleuropneumonia.

Clinical Symptoms:

1-The symptoms appear within 1-3 weeks (after feedlots beginning and after shipping).

2-Sudden death in fatal course due to sever hypoxia & Asphexia.

3-Fever, anorexia, emaciation, dyspnoea and accelerated respiration.

4-Extenssion of head and neck with each respiration.

5-Diseased animal is separated from the flock and recline.

6-Drooping of ears and dull appearance.

7-Muco purulent nasal discharge and lacremation.

8-Cough with the temperature of 41-42C, difficult breathing.

9-About 30% of the lung become affected within one week after beginning the disease.

10-By auscultation: in congestion and gray hepatizatin stage we hear "harsh exaggerated vesicular sound" while in red hepatization we found dul sound due to considledation of the lung in these stage.

11-Some lambs may be lame due to arthritis (mycoplasma synovae).

12-Morbidity up to 50%, mortality up to 10%.

13-Course of early fatal case 2-3 days but non-fatal cases recover after

14-20 days.

14-All flock may become infected within one months.

Complications:

1-The serofibrinous excudate may leads to adhesion of:

-Lung lobs together. -Lung and pleural sac.

-Lung with the chest wall. -Pleural and pericardial sac.

2-Pleuritis which characterized frictional sound.

3-Pericarditis due to extension of infection to the heart.

4-Ventericular hypertophy due to resistance of pulmonancy circulation w` may leads to CHF.

Post-mortem lesions:

1-Stage Of Congestion:

-Conested or cyansed lung tissues.

-Large amount of exudate present in the pleural or pericardial sac [complicated cases]

2-Stage of Red Hepatization:

-Small, and deep red colourd exudate.

-Solid tissue of lung in consistency.

-Complications mentioned before.

3-Arthritis in some animals (due to infection by Mycoplasma synovae).

4-Variable degree of consolidation of venteral part of lung especially a pical and cardiac lobes.

5-In Gray Hepatization:

-The lung is purpel-gray, firm and rigid.

-Abundant serum and fibrine in the pleural.

6-In Stage Of Resolution:

-Lung is pink, flexible and moderately aireated.

-And has lesser amount of serum and fibrin in pleural cavity.

7-All stages may appear in the same lung.

8-At the junction between the pneumonic tissue and healthy tissue emphysema and atelectasis may be present [lobular pneumonia].

9-Regional lymph nodes are swollen and hyperemic (mediastinal and bronchial lymph nodes).

10-Peticheal and echymotic haemorrhage are widely distributed over serosal surface specially lungs and heart.

11-The bronchi, tachea, larynx, pharynx, nasal cavity and para naasal sinuses usually reveal catarrhal inflammation.

12-Blood clots spots specially in pasteurele haemolytica infection.

Histopathological Picture: according to stages of the disease.

1-Initial stage: odema, congestion, and proliforation of septal and epithelial cells predominant.

2- During red consolidation:

-Fibrinous exudate fill some alveoli. -Leuocytes obscure the fibrin.

-Thrombi form in lymphatic (lymph vs.).

3-During resolution → partially or fully lysed fibrin is replaced with

macrophages.

4-During early stage pasteurella and chlamydia can be detected in Giemsa- stained smears and sections.

Diagnosis:

1-History, clinical symptoms, postmortem and histopathological forms.

2-Differential diagnosis-parasetic pneumonia: septicemic pasteurellosis, polyarthritis.

Treatment And Control:

1-Isolation of diseased animal.

2-Long-acting oxytetracycline with sulfa are the best drugs of choice.

R1- Allamycin L.A. 1ml/10kg Bwt. R1- sulph demidin sool. 33,1/3%.

R1- Terranrycin L.A. 1ml/10kg Bwt. R1- unisulfa inj.

R1- Advocin. L.A. 1ml/10kg Bwt. R1-

3-In out breake of pneumonia total flock therapy via the water or feed with sulfonamide (1mg/kg) or oxytetracyeline (12mg/kg) should be considered.

4-Bronchodilator As; R1 Mino phyllin – etaphyllin, thiophyllin …etc.

5-Preventive and mangmental measures to avoid the stress factors.

6-Vaccinal programes for the viral diseases.

As Adenovirus, respiratory cynciytial virus, parainfleeunza type3:

6-Ovine Adenovirus (OAV)

(A) Initial phase:

1-Serous nasal discharge & sneezing.

2-Coughing-Anorexia and conjuncitivitis & lacremation.

3-Forced respiration usually accempained by diarrhoea.

4-Lambs of 2-4 months and weaned lambs during feeding period are highly susceptible.

5-Swelling of retro pharyngeal lymph node & submandibular lymph node.

(B) Chronic form:

1-Mucopurelunt nasal discharge.

2-Diarrhoea 1-2 weeks of duration.

3-Spentenous cough.

4-Persistent nephritis and weak body condition.

PM lesion:

(1)- Acute phase:

1-Rhinitis-acute catarrhal pneumenia.

2-Enteritis may be present.

3-Enlargement of retro pharyngeal and mediastinal l.n.

(2) Chronic phase:

1-Chronic intera lobular inter stitial pneumonia.

2-Peri bronchitis and preoliferation of peribronechial lymphoid fllicles.

7-Mycoplasma Pneumonia & Chlamydial Pneumonia"

1-Mycoplasma ovipneumonia has been isolated from the lung of healthy and pneumonic sheep often in combination with other microorganisms. However, the role of mycoplasma in the etiology of pneumonia has not been established.

2-Chlamydia psittaci is considered by some investigators to be a primary agent of ovine pneumonia.

3-Chlamydia leads to:Polyarthritis, conjunctivitis. Abortion, pneumonia.

4-Penicillin, sulfa, and tetracycline preparations are effective against chlamydia.

Chapter No (6)

Skin Diseases

1-Photosensitization

2-Eczyma

3-Dermatitia

4-Acne

5-Pityriasis

6-Parakeratosis

7-Hyperkeratosis

8-Urticaria

9-Alopicia

10Anasarca

11-Angioneuretic odema

1-Photosensitization

Synonoms:-1-Big head 2-Swollen head 3-Clover disease

Definition: It's acute dermatitis in non-pigmented sheep characterized by irritation, odema, and anasarca (generalized s/c. odema)]and necrosis of ears, eyelids, lips, face, vulva and cornet and caused by interaction of plant-formed pigments and sunlight.

Occurrence: 1-Occur in all sheep producing countries

2-All breeds, ages and sexes of non-pigmented sheep.

Etiology: ***1-Primary Photosensitization:***

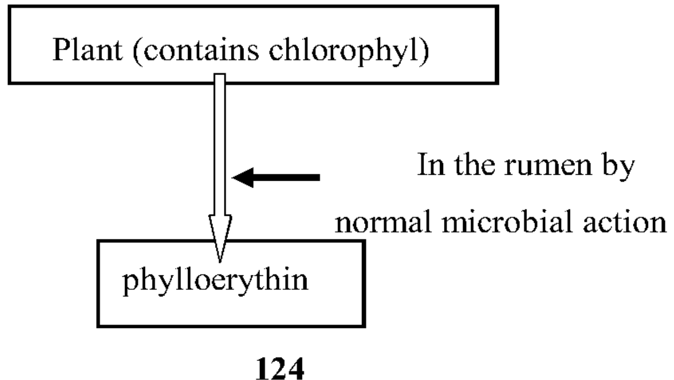
-Due to feeding on uncommonly plants contain photodynamic pigment as saponine or mycotoxin……etc. -Some drugs as phenothiazine

2-Secondary Photosensitization or (hepatogenous)

-Due to effect of pyrolizidine.

-Alkaloid containing plants (rare in sheep) or diseased liver.

Pathogenesis1-Normal metabolism of plant,producing pigment in ruminant.



2-Phylloerythin absorbed by portal circulation then to liver then → Reach the bill for excretion and consequently the peripheral blood circulation is free from phylloerythrin.

3-Diseased liver or unhealthy bill circulation. So the animal unable to excrete the pigment → and consequently it's accumulated in the blood circulation. → causing generalized phylloerthemia.

4-Phylloerythrin usually presente in inactive form and when the skine exposed to the sunlight the phyloerythirin is converted into active form (antigen) casing antigen antibody reacation and release of histamine

5- In the presence of protein molecules ,aminoacids and oxyegen it leads to formation of → Toxic compounds that leading to → increase the permeability of the blood capillieries to plasma protein and consequantly leads to force the water into the affected tissue (increase the hydrostatic pressure of the blood vessels) → Oedema and necrosis.

6-Death due to secondary bacterial invasion, liver dysfunction or inability to feed and unthriftness.

N.B. Sheep that possesing haemoglobin A or AB are resiste to such conditions while that possesing haemoglobin B are highly susciptible within unknown interpretation.

Clinical Symptoms:

1-The time from ingestion of plants containing photodynamic pigment and appearance of clinical signs varies from several hours to 10 days.

2-The disease begins with:

-Erythema. -Photophobia

-Irritation of the: -Lips-vulva, face,Cornea, cornet and Eyelids.

3-Rubbing and Kicking the affected partes.

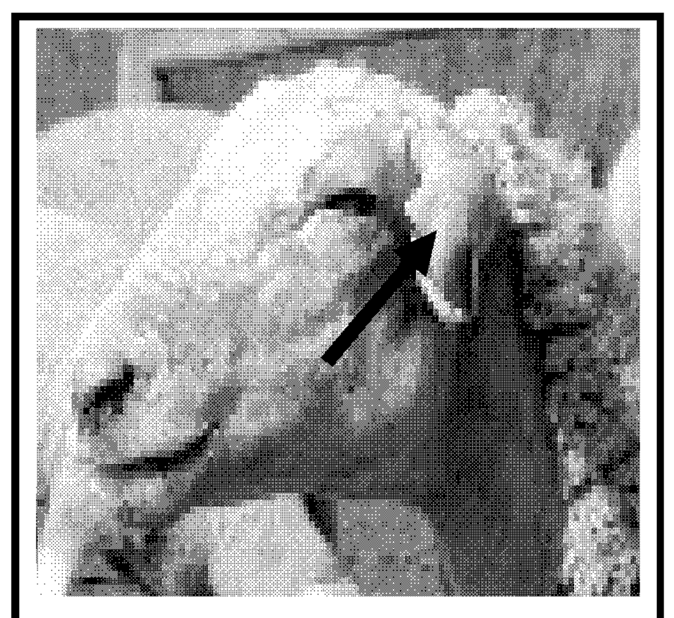
***4-Facial odema and in this stage there are*:**

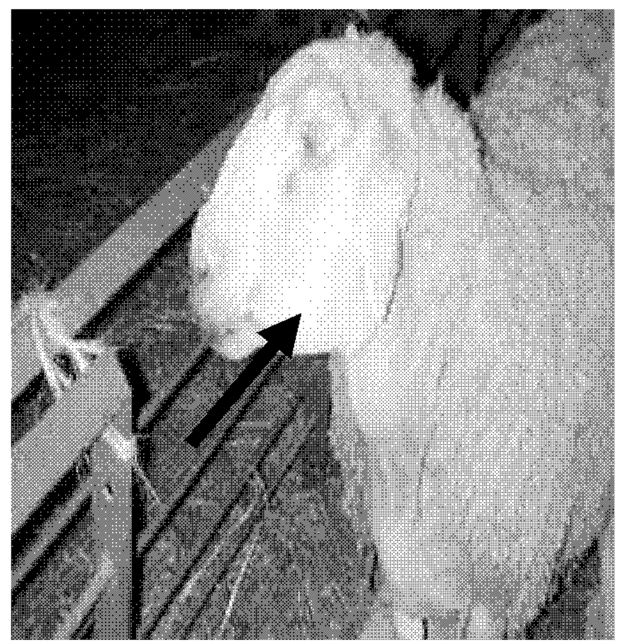
-Thickened lips and immobilized. -Constricted and swollen nostril.

-Dropping of heavy ears. -Animal suffering from dyspnoea and a norexia.

5-The diseased animal is separated from the flock, depressed and decline.

6-The animal that in convalescent may show dry gangrene and sloughing of the skin specially in the ears.





Photosensitization in sheep

7-Morbidity up to 80% of the flock, mortality up to 20% of affected animals while the course from few hours to weeks.

8-Icteric mucous membrane may occure.

PM lesions:- 1-Facial, vulvar, and pedal skin commonely is odematous.

2-Generalized icteric carcase as in case of secondary photo sensitization. (hepatogenic photosensetization)

3-Liver lesions may present as cirrhosis or bill duct obliteration.

Diagnosis:- *1-Case history of :-*

-Feeding on uncommonly plant. -Non-pigmented sheep common affected.

-Administration of some drugs. *2- Clinical symptoms and PM lesions*

3-Detection of the level of photo dynamic pigment as phylloenythin .

4-Differential diagnosis:

-Either due to primary or secondary photosensitization.

-Liver diseases as aflatoxicosis, liver abscesses or corpulmenale.

-Other skin disease.

Prevention And Treatment:- 1-Avoid grazing on pasture that containing photosensitizing agents. 2-Removing the plant that contain photodynamic pigmente from the diet. 3-Rectal enema of affected animal.

4-Anti histamenic (R/Avil- R/Vetibenzamine, R/Tavagyle…..etc.)

5-Anti-inflammatory R1-Dexamethazen-Solucertife……etc.

6-Dieuretic. 7-Cold bath and supplementation of palatable food.

8-Sheep with chronic liver disease rarely recover.

9-Antimicrobial drugs to avoid the secondary bacterial infection.

2-Eczyma or False Mange

Definition:- Eczyma means inflammatory reaction of the epidermal cells due to presence of substances which sensitize or stimulate the inflammatory process in the skin and such substance may be internal or external.

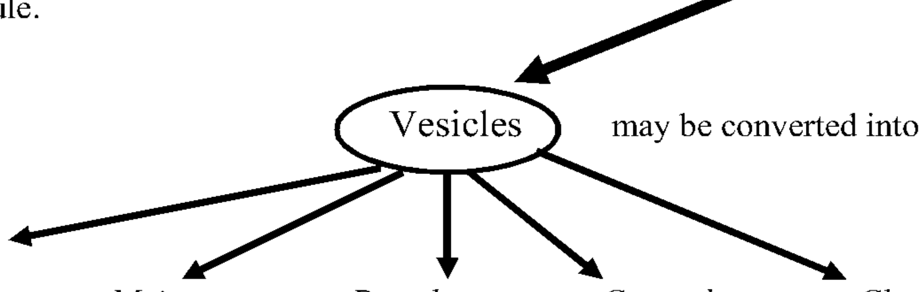
Etiology:- -The main cause is presence of allergin which may be:

(A) External or (Exegonous)- 1-Using of strong or concentrated disinfectants or antiseptics solutions on the skin. 2-Administration of Iodide, mercuric or arsenic drugs.

3-External parasites (mange). 4-Prolonged wetting of the skin.

(B) Internal or (indigenous):-1-As absorption of toxic hepatitis, nephritis, constipation or ingestion of certain protein (the allergin is absorbed and reach the skin through blood circulation).

Pathogenesis:-The allergic or inflammatory reaction between the skin and allergin (either external or internal allergin) leading to → Erythema then to Papiule.



Dry eczyma Moist eczyma Pustular eczyma Crusted eczyma Chronic eczyma

***1-Dry Eczyma*:** → Characterized by: *Absorption of the contents of

vesicles *Ulceration and scare formation.

2-Moist or wet Eczyma: Characterized by: *Rupture of vesicles.

*Oozing the contents of the vesicles on the surrounding skin.

3-Pustular Eczyma: Characterized by:

*The vesicles are invaded by pyogenic microorganisms.

*Formation of pustules.

4-Crust Eczyma: Characterized by:

*Dryness of the vesicles. *Crust formation.

5-Chronic Eczyma: Characterized by: → occurs when any forms of that mentioned before is persist more 3 weeks, it called chronic eczyma.

Clinical Symptoms:

1-Erythema, itching and rubbing themselves similar to manage ,so it called "***false mange***".

2-Symptoms vary according to the form of eczyma that mentioned before in pathogenesis. 3-Morbidity up to 50%.

Diagnosis: 1-History and clinical symptoms.

2-Skin scraping for differentiate with mange.

Treatment: **1-*Hygienic treatment***: by avoiding the source of allergin either internal or external parasites……etc.

2-Local treatment:

*In case of moist or wet eczyme we can use dust powder over skin as:

R/Tanic acid 5gm + salyselic acid 5gm.

===
Or R/Zinc oxid 10gm + powder talce 10 gm.

*R/Sulpher powder 5gm + Alcohol sol. 40 ml +Formalin sol 5ml With Olive oil Up to 100ml and then used as skin application.

*Anaesthetic skin ointment as legnocain ointment

3-Systemic Treatments:

-Anti histaminic, tonics and antibiotics.

-Treatment of hepatitis, nephritis if suspected.

3- Dermatitis

Definitions:- Dermatitis means that inflammation of the skin including epidemis, dermis and may include blood vs& lymphatices, and it may be:

1-Bacterial dermatitis: due to staphylococcus aureus.

-Corynibacterium pyogen. -Streptcoccus spp.

-Straweberry footrot-dermatophilus pedis.

2-Viral dermatitis: as in case of :-

-Pox - Foot and mouth disease(FMD). -Ulcerative dermatosis

-Contagious ecthyma -Blue tongue disease.

3-Mycotic dermatitis:

-As in ring worm. -Dermatophylus congelensis [lumpy wool]

4-Metazon dermatitis: *as* -Sarcoptic and sorpotic mange.

-Photosensitization (primary or secondary).

(6) Chemical dermatitis:*-As due to strong acid as H_2So_4, or HcL.

*-Aresenic preparation either local or parental with high concentration.

(7) Allergic dermatitis as:- -Certain oil, soap or other antigen. (External or internal antigen) as mentioned before in eczyma.

(8)-Nutritional dermatitis: As

-Zinc deficiency -Vit-A deficiency. Or vit- B deficiency.

Clinical Symptoms:- 1-The affected skin area first show erythema and hotness.

2-The subsequent stages varies according to the type and the severity of the causative agent.

3-There may be development of vesicular lesions or dffuse weeping.

4-Odema of skin and s/c tissue may occur in sever cases.

5-The next stage may be healing stage of scab formation or if the injuries are more sever there may be increased or even gangrene in the affected skin area.

6-Spread of infection to s/c tissue may leads to cellulitis or phlegmonous lesion or pyoderma (suppurative lesions).

7-A systemic reaction is likely to occur when the affected area is extensive.

8-Shoke with peripheral cericulatory failure, may be present in early stage.

9-Toxaemia due to absorption of tissue break down products or septicemia due to invasion via unprotected tissues may occur in later stage.

10-In Ovine atopic dermatitis:

(A)-Only woolless partes of the skin are affected by symmetrical erythema.

(B)-Only occosional sheep in the flock are affected usually in each summer with remission during the winter months. (C)-Alopecia.

11-In case of oxidative dermatitis of goat :-

*All ages and both sexes of pygmy goat may be affected and characterized by hair loss and scaling and crusting around the eyes, lips and chin, ears, pool, perenium and venteral abdomen are affected.

Diagnosis:-

1-Clinical features and history.

2-Skin biopsy and skin scarping to detect bacterial or parasitc dematitis receptively.

Treatment:-

1-Attack the main cause of the disease

2-Application of local antibiotics or antifungl.

3- Administeration of Anti histaminic drugs.

4-Anaesthetic ointment when pain or itching is severe.

5-Providing of vit-B in the food specialy in case of vit.B deficiency.

6- Application of Astringent ointment

7-If shock present fluid therapy should be administered.

8-Protein supplementation in diet for enhancing repairing of skin lesion.

9-Vaccination againest viral causes as pox.

4-"Acne" & "Folliculits"or Sycosis

Definition:- Acne means that infection of the sebaeceous gland or hair follicle by acne bacilli or by any other suppuorative microorganism as staph. (usually termed sycosis).

Etiology And Predisposing Factors: 1-Blockage of sebaecious gland ducts or desquamated epithelium or dilatation of such ducts may leads to reach the microorganisms to the hair follicles.

2-Infection usually occurs by acne bacilli or staphylococcus usually associated with mange as a predisposing factors (usally demodectic mange).

Clinical Symptoms: 1-Presence of painfull papeules at the base of the hair, this papules may become pusteules due to invasion of supuorative microorganisms. 2-Under pressure the pusteules may be ruptured and spread the Infection to the surrounding tissues with itching.

3- Alopecia at the affected area due to shedding of the hair.

Diagnosis:- **1**-Skine symptoms and swabbing for examination.

3-Differential diagnosis with impetigo.

Treatment:- **1**-Isolation of the diseased animal.

2-Application of local antiseptics on the affected area. R. /Betadin or Savolen.

3-Antibiotices local and systemic. (For 1 month). R/ Terramycin skin Oint

R/ Teracortril R/ Terracortrin R/Gentamycin Oint.

4-Trimethoprime with sulphathiazine preparations gives good results.

5-Pityriasis or Dandruff

Definition: -

It's a diseased condition characterized by bran like scales on the skin specially in the buffalo.

Etiology:-

1- Nutritional deficiency as vitamin-A deficiency.

2-Nicotenic acid and Riboflavin deficiency.

3-Parasitic as mange on other external parasites.

4-Infectious as: Ring worm.

Symptom:-

1-Scales with itching in infectious or parasitic type.

2- Scales with out itching in Nutritional type.

Diagnosis:-

-History and clinical symptoms.

-Skin scraping and biopsy.

Treatment:-

Removing the scales and removing the causes.

6-Parakeratosis

Definition:-

* Parakeratosis means that inadequate keratinization of epithelial layer of the skin which mainly due to zinc deficiency.

Clinical Symptoms:-

1-Firstly appearance of red area converted into gray colored area.

2-The lesions appeared an the thigh specially at area of the joints in the form of alopecia and the skin appeared as raw skin.

3-The lesions may be cracked in network form, and secondary bacterial invasion may occur.

4-See also zinc deficiency.

Diagnosis:

1-Clinical symptoms.

2-Detection of zinc level (0.02-0.04 mg %).

Treatment:

1-Washing the skin by soapy solution. A white lotion [2gm zinc sulfate + lead acetate 3gm + 5oml distilled water] or salicylic acid ointment as- astringent.

2-Zinc oxide 1/2 gm. orally. everyday for 3weeks or addition of zinc sulfate on the ration (see also. Zinc deficiency).

7- Hyperkeratosis

Definition: -

It is extensive keratinization of epithelial layer of the skin which may be:

*Generalized as: in arsenic poisoning.

*Local As in elbow area (in buffaloes) or at saddle in equine due to such.

Area is fractionated usually with rough abject.

Symptoms:

1-Area of alopecia and increase the thickness of the skin.

2-Scale formation and dryness of the skin.

3-Cracks and fissures in network from and secondary invasion may occur.

Diagnosis

:Clinical symptoms and skin scraping and biopsy.

Treatment:

Washing the skin by soapy water.

1- Removing the excessive keratinized layer by astringent as white lotion (zinc sulfate 2gm + lead acetate 3gm + 5o ml D.W)

8- Urticaria

Definition: Urticaria means it is allergic condition of the skin characterized by formation of wheels on the skin due to excessive release of histamine.

Etiology: **A- Primary causes:**

1- Feeding on feed rich in "CHO" and protein with inadequate exercise
2- Recent sudden change in diet.
3- Injection of some drugs as penicillin.
4-Feeding an unusual food.
5-Milk allergy in dried animal.
6-Constipation with gastroenteritis.
7-Insecte bites ..

B- Secondary Causes.

1-mainly due to inhalation of allergen or respiratory tract infection.
2-Destruction of cells and releasing of histamine.

Pathogenesis:

-Allergen [antigen] + antibody reaction → Release of histamine
→ Vasodilatation of the peripheral blood vs. that leading to → Erythema.
-In crease the permeability of capillaries to plasma causing odema and wheels.

Clinical Symptoms: Restlessness and the lesions appear as circumscribed

elevated area of the skin 1/2-5 cm in diameter in different areas of the body.

1-Erythema and itching specially in insect pits.

2-Diarrhoea or fever may occur.

3-Odema of ears, muzzle and vulva.

4-Symptoms may disappear within 3-4 day or after lowering the level of histamine to normal level.

Diagnosis:

1-History and symptoms.

2-Blood examination to detect eosinophilia.

Treatment:

1-Purgative or gastric lavage to get ridding of noxious food. By using of R1 Mag. sulfate or vegetable al or mineral oil.

2-Antihistaminic as: -   Avil - Allecure - Tavagyl ....etc.

3-Adrenaline as vasoconstrictor up to 5cm I M or S/C. 1%.

4-White lotion (2gm zinc sulfate + 3 gm lead acetate + 5 ml D.W).

5-Calomina lotion.

6-Sodium bicarbonate. As a washing solution.

7-Calcium borogluconate or any other calcium preparation because of.

*Calcium has anti-histaminic effects as it affection on the mast cells.

*Has vasoconstrictor effect as it increase the viscosity of the blood.

9- Alopecia

Definition: Alopecia means that loss of hair (in goat) or wool in sheep and it may be localized or generalized.

Etiology: 1-Long period of starvation leads to the sheep begin to eat the wool of each other specially at the flank region.

2-Overeating may leads to shedding of wool or hair.

3-Recovery from high fever may leads to hair loss.

4-Ring worm or mite infestation (mange) or sever wounds.

5-Nutritional as vit. A, zinc, cobalt, Iodine deficiency.

6-Viral infection of dam during pregnancy.

7-Inability of hair follicles to produce. Hair either due to hypo-thyrodism or infection of hair follicles itself. (Acne or follecultis).

8-Deep wounds which may destroy the hair follicles.

9-Sever rubbing or itching of skin. 10-Metabolic dermatitis.

11-Some physical or chemical agents [Hot, Arsenic, Hcl- etc.]

So, Alopecia may be:

- Nutritional - Infectious. (Acne) -Metabolic. - Physical.
- Chemical. - Endocrinological. -Manegmental - Hormonal

Clinical Symptoms:

1-Absence of wool or hair coat and consequently the animal become more susceptible to the external environmental conditions.

2-Alopicia may associate with itching or pruritis as in mange.

3-Crumplign loss of the wool.

4-Broken wool and hair and the stumps of the wool and hair may be observed at the site of alopecia.

Diagnosis: **1-**Clinical symptoms and History to differentiate between nutritional and other types of alopecia.

2-Skin scraping if mange or ring worm is suspected

3-Blood examination to detect the level of zinc, cobalt & vit-A .

Treatment: Depend mainly upon. Diagnostic type of alopecia according to it's cause.

10- Anasarca

Definition: Anasarca means that it is localized or diffuse swelling (edema) under the skin and it's considered as part of generalized edema.

Etiology

A- Circulatory disturbances [vascular resistance]

1-Congestive heart Failure.

2-Enlargement of large blood vessel due to high pressure.

3-Closure of large blood vessel from external due to tumor specially mediastinal lymphasarcema.

4-Absence of some lymph node or lymphatic

B- Hypoproteinaemia:-1-Liver diseases that lead to in adequate production of

Albumin that affecting on osmolality of plasma.

2-In adequate protein intake in the diet.

3-Heavy infestation of gastrointestinal warms. Specially fascioliasis.

4-Renal damage accompanied with loss of protein in urine (proteinuria).

C- Disturbance in blood vessels. (damage of bl. vs)

1-Purpura haemorrhagica. 2-Hypovitaminosis-A.

Clinical Symptoms:

1-Painlless localized diffuses subcutaneous edema.

2-The swelling is painful in inflammation process as in infection by clostridium. 3-Pits on palpation of edema.

4-Odema usually in the ventral aspect of the trunk.

Diagnosis: 1-History and clinical symptoms.

2-Blood examination to detect total protein & albumin.

3-Fecal examination to detect GIT Nematodes.

Treatment: -Treatment the causative agents with diuretics.

11-Angioneuretic edema "Angioedema"

Definition: Sudden appearance of transient skin edema due to allergen i.e. antigen antibody reaction that leading to histamine release and oozing of plasma outside the blood vs. and causing the edema.

Etiology: **1-**Exogenous or endogenous allergen [see urticaria]

2-Occurred mostly in sheep and cattle during pasture specially in the

flowering season i.e. the allergen is of plant origin.

3-Fish meal may induce the disease.

Pathogenesis:

Antigen – antibody reaction → Histamin release → vasculor damage →Odema.

Clinical Symptoms:

A- Face:

1-Swelling of lips, eyelids and conjunctiva.

2-Lacremation, salivation and nasal discharge.

3-Protrusion of 3rd. eye lid.

4-Rupping the head against object.

B- Udder and perineum: Asymmetrical edema of udder & vulva.

Diagnosis:

Depend upon.

1-Clinical examination.

2-Season of the disease.

3-Type of food.

Treatment:

1-Isolation of the animal form the area of grazing.

2-Supplementation by dry ration 1-2 weeks.

3-Laxative and purgative to remove the excess of allergen.

4-Adrenaline. 3-5ml 1% to make. Vasoconstriction.

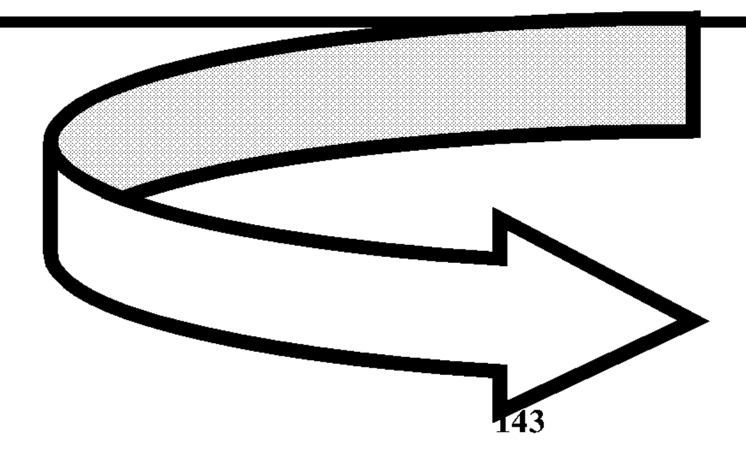
5-Anti-histamenic (Tavagyl-Avil-Allecure- …..etc.).

6-Diuretics if edema is sever.

Part (II)

Large Ruminant Medicine

(Cows and Buffalos)



Chapter No.(1)

Digestive System

1-Disease of the muzzle
2-Stomatitis
3-Diseases of the teeth
4-Parotitis
5-Pharyngitis
6-Pharyngeal paralysis
7-Esophagitis
8-Esophegeal Obstruction
9-Introduction to stomach diseases
10-Simple indigestion
11-Impaction
12-Bloat
13-Trumatic reticulopertonitis
14-Vagus Indigestion
15-Left and right displacement of abomasums
16-Peritonitis
17- Liver diseases

A-Diseases Of The Buccal Cavity

1- Diseases of the muzzle. **2-Stomatitis.**

3- Diseases of the teeth. **4-Parotitis.**

1-Diseases Of The Muzzle

1-The congenital defect of the harelip may be contagious with a cleft palate.

2-Sever dermatitis with scab formation, development of fissures and sloughing and gangrenes of the skin of the muzzle are more common in the cattle.

3-The diseases in which these lesions occur are:

a) Photosensitization

b) Bovine viral diarrhea

c) Bovine malignant catarrh

d) Render pest.

2- Stomatitis

Definition:- Stomatitis is inflammation of the oral mucous membrane including: A-Glositis (inflammation of the lingual mucosa).

B-Gingivitis (inflammation of the mucosa of the gums).

C-Palatitis or lamps (inflammation of the palate) and stomatitis characterized clinically by excessive salivation, anorexia and smacking of the lips.

Stomatitis usually is accompaniment of systemic disease.

Etiology: May be: (I) Physical (II) Chemical (III) Infectious (more common) (IV) Nutritional (experimentally).

(I) Physical agents:

1-Trauma while dosing by stomach tube. 2-Laceration of the tongue.

3-Foreign body injuries. 4-Malocculoded or sharp teeth.

5-Sharp awns or spines or plants (more common in the gums).

6-Drinking of so hot or so cold water or fluids.

7-Fracture of the mandibles due to strong trauma from outside.

(II) Chemical agents:

1-Irritant drugs administered in high concentration such as: chloral hydrate.

2-Counter irritant on the skin and left unprotected then licked by the animal as mercury and cantharides compounds. Or Iodine.

3-Irritant substances as acid or alkaline taken by mistake.

4-As a complication of systemic cases as uremia.

5-Manifestation of some poisoning condition as mercury poisoning (chronic).

(III) Infectious agents:

A-Viral causes as in case of:

1- FMD. 2- Vesicular stomatitis.

3- Cattle plague 4- Malignant Catarrhal Fever (MCF).

5- Infectious Bovine Rhinotracheaitis(IBR)

6-Bovine Viral Diarrhea -Mucosal Disease complex (BV.D-MD)

B-Bacterial causes as in case of :

1-Actinobacillosis (wooden tongue) caused by Actinobacilus ligneresi.

2-Actnomycosis (lumpy Jaw) caused by Actinomyses bovis.

3-Oral necrobacillosis (fusibacterium necrophorus).

(IV)- Mycotic stomatitis:

May occur due to Infection by Candida albicanis or Aspergillus spp.

(V)- Nutritional causes:

-Only under experimental conditions found that Vit. A deficiency may lead to oral lesions simillar to that of stomatitis.

Pathogenesis:-

1-The causative agents of stomatitis may be applied directly on the mucosa or get entrance to the mucosa through abrasions in it and in such conditions it considered as primary stomatitis.

2-The causative agents may be localized in the mucosa through the

blood stream as in case of viremia or bacteremia or uremia and such conditions considered as secondary stomatitis.

3-The symptoms and lesions vary according to the causative agents and degree of inflammation.

Clinical Findings:-

A) General or systemic symptoms:

1-Fever in case of infectious agents.

2-Anorexia with profuse salivation

3-Impaired mastication, prehension with smacking of the lips.

4-The saliva may contain pus or shreds of epithelium.

5-Fetid odor of the mouth occurs only if the lesions are invaded by bacteria → with frothy saliva at the mouth commissures.

6-Enlargement of the localized lymph node.

7-Swelling of the face if the lesions extended to soft tissues causing cellulites or phlegmon as in case of actinomycosis (lumpy Jaw) or actinobacillosis(wooden tongue).

8-Increase the desire for water and the animal very nervous during the manipulation or examination of the mouth.

B) Local lesiones:

1-Vesicular lesion:

Usually thin – walled vesicles 1-2 cm in diameter filled with clear serous fluid, this vesicles rupture readily to leave sharp-edged shallow ulcers (vesicles rupture → shallow ulcers).

2-Catarrhal stomatitis:

Is manifested by a diffuse inflammation of the oral mucosa and is commonly the result of direct injury by chemical or physical agents.

3-Lacerated lesions:

On the lips and tongue that indicated to traumatic cause.

4-Proliferative stomatitis:

Small (less than 1 cm diameter), swollen congested lesions on the tongue, oral mucosa and palates which undergo ulceration within 3 days (usually occurred in viral infection).

5-Papular stomatitis:

Raised reddish papules (0.5-1 cm in diameter) found on the oral mucosa and muzzle or may be present on the nostrils (it's also may considered as a form of viral infection and it's associated with systemic reactions). (Papules → vesicles → ulcers or pustules).

6-Pin pointed ulcers:

-Follows the papules and it occurs usually in cattle plague or BVD.

-These ulcers coalesced together to form denuded ulcers.

-These lesions may be present: - *All over the mouth cavity.

* Pharynx and esophagus. * Abdominal segments of the G.I.T.

-There is no vesicles formation in this form and the body temperature may reach 40-41°C.

7-Necrotic stomatitis:

The lesions found on the mucosa of cheek, also may be found on

pharynx and larynx, the cheeks looked as swollen cheeked appearance.

8-Actinobacillosis: (wooden tongue):-

Characterized by presence of abscess like lesions on the tongue and may be on the lymph node.

9-Oozing of the food in-between the lips leading to staining of the hair around the mouth giving an appearance similar to that of the tobacco-chewer (specially in sheep).

10-Presence of diphtheric membrane specially in case of uremia.

11-Mycotic stomatitis characterized by heavy white and velvety deposits with less obvious inflammation or damage of the mucosa with history of long period of antibiotic therapy.

Diagnosis:-

1-History : A-Depend upon presence or absence of fever.

B-Duration of the disease.

C-its development of the lesions.

2- Clinical symptoms. 3- Isolation and culture from the lesions.

4- Serological tests: as in case of viral disease.

Treatment:-

A) Hygienic : 1-Isolation of the affected animal specially if infectious diseases are suspected. 2-Fresh clean water supply.

B) Medicated treatments:

(1) General treatment:

[I] Systemic antibiotics:

1-Penicillin and Tetracycline are effective in necrotic form. Or in viral form to avoid the secondary bacterial invasion. 2-3 millions I.U/day I.M. of Penicillin or 2-3 gm Tetracycline / day I.V. or I.M. for 5-7 days.

2-Sulphathiazine 120 gm orally then half of the dose every 12 hours orally for 5-7 days.

3-Vaccination program against the viral diseases.

4-Using of Anti-sera.

5-Iodides preparations in case of actinomycosis and actinobacillosis (pot. Iodine – sod. Iodide). 9 gm/100 kg B.wt. in 10-20% solution I.V. (i.e. 50 ml/100 kg 20%) slowly (also organic iodide can be given orally).

[II] Local treatment:

A-Anti-Septic solutions to wash the oral cavity as:

1-Pot-permanganate 1: 50000 2-Boric acid 2%

3-Acriflavin 2% 4-Borax 1-2%.

5-Tannic acid 2% 6-Cupper sulphate 2%.

B-Cauterizing agents to swab the lesions as:

(1) Silver nitrate 1% (2) Sulphonamide in glycerin 0.5-1%

(3) tri-Iodine 1-2%.

C-Radiation therapy: used for treatment of actinomycosis and actinobacillosis (Never to be used before 30 days elapse after iodide therapy).

3-Diseases Of The Teeth

(1) Congenital defects:

(A)It may be inherited.

(B)Malocclusion of teeth to degree enough to interfere with mastication and prehension.

(C)Red brown staining of inherited porphyrinuria in cattle.

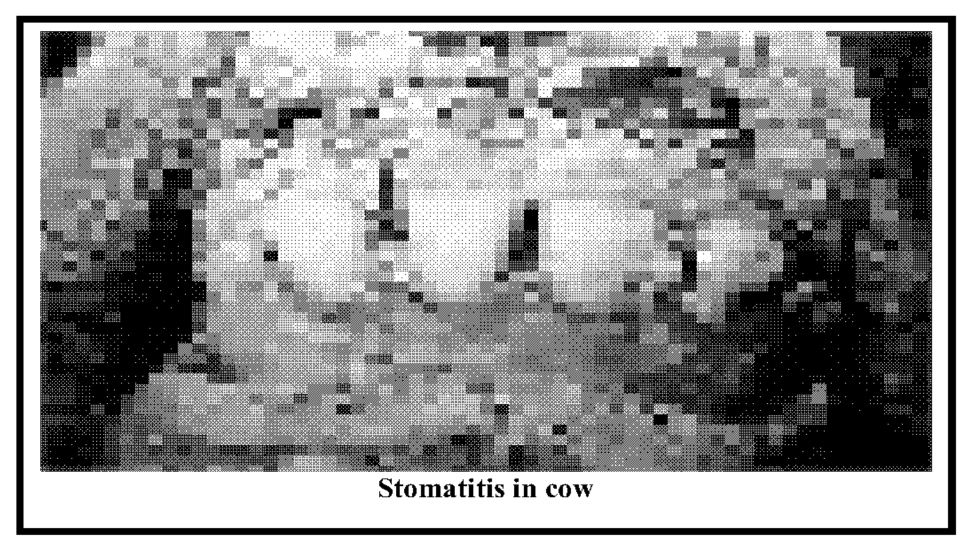
(D)Defected enamel formation as all teeth combination with excessive mobility of the joints in inherited defect of collagen metabolism in Holstein and Frisian cattle identified by osteogenesis imperfectia.

(2) Enemal hypoplasia:

That condition is recorded in experimental infection of helminthes parasites

(3) Periodentosis:

See sheep and goat medicine department.



Stomatitis in cow

4-Parotitis

Definition: Parotitis means inflammation of any of the salivary gland (usually occur in sporadic form).

Etiology:- **1**-There is non-specific causes but invasion of the salivary ducts by awns may lead to parotitis.

2-Stomatitis and hypovitaminosis-A may be considered as predisposing factors to Parotitis.

3-Secondary bacterial invasion of the salivary gland or ducts through the blood stream.

4-Stone in the salivary duct (sialolithiasis) predispose to parotitis

5-Local suppurative lesions are caused usually by penetrating wounds or extension from pharyngeal cellulitis or lymph node abscesses.

Types:

1-Paranchymatous parotitis → means that all salivary gland tissues are affected or inflamed.

2-Local supurative lesions → mean local areas only affected more dangerous than paranchymatous because it's more persistent and may lead to fistula.

Pathogenesis: The inflammation and loss of functions usually concerned with the affected glands only.

Clinical symptoms:

A) Early stage :

(1)Diffused enlargement of the gland.

(2)Warmth and pain in palpation which may interfere with mastication and swallowing.

(3)Abnormal carriage of the head and the animal show the sings of the pain if try to move the head.

B) In sever case:

- Diffuse paranchymatous parotitis is subsided within few days by local and systemic treatment while local suppurative lesions may lead to salivary fistula.

Diagnosis: (A)History and clinical symptoms.

(B)**Differential diagnosis:** 1-Acute phlegmonus inflammation of the throat which is common in the cattle which characterized by:

-Sudden onset of fever, toxemia and death. - More diffuse swelling.

-Pronounced obstruction to swallowing and respiration.

2-Lymphadenitis or lymphoma.

3-Goiter or any swelling in the pharyngeal region.

Treatment:

1-Systemic antibiotics or sulphenamide specially in acute stage.

2-Providing of vitamin- A in case of vitamin- A deficiency.

3-Surgical interference in case of salivary fistula.

4-Dealing with stomatitis if it's suspected.

B-Diseases Of The Pharynx And Esophagus

1-Pharyngitis. 2- Pharyngeal obstruction.

3-Pharyngeal paralysis. 4-Esophagitis.

5-Esophegeal obstruction (choke).

(1) Pharyngitis

Definition:

Pharyngitis means that inflammation of the pharynx and it's clinically characterized by painful swelling, coughing, and variable appetite and in sever cases there is regurgitation of the food and/or water through the nostrils.

Etiology:

A) Physical and chemical agents:

1-Trauma during use of the tracheal intubation or balling or derching gun.

2-Accidental administration or ingestion of irritant or so hot or so cold substances.

3-Foreign body lodged in the pharynx.

B) Infectious agents:

1-Nicro-bacillosis or actino bacillosis (wooden tangue).

2-Infectious bovine Rhinotracheaitis (IBR).

3-Fusobacterium necropherous.

4-Bovine viral diarrhea – mucosal disease complex (BVD-MD complex).

C) *Extension from:*

1-Stamatitis or parotitis.

2-From lymphadenitis (inflamation of the pharyngeal or retropharyngeal lymph node).

3-Inter-mandibullar cellulitis or pharyngeal phlegmen.

Pathogenesis:

A-Physical or chemical agents: lead to → laceration of the pharyngeal mucosa and esophagus. → Accumulation of the ruminal ingesta in such wounds (during rumination)or in the divirticulae on both side of glottis → (cellulitis and pharyngitis).

B-Infectious agents: according to the pathogenesis of each infectious disease the microorganism may reach the pharynx directly or through the blood or lymph stream then causing the inflammatory action (pharyngitis).

Clinical symptoms:

1-The animal reluctant to eat or drink.

2-The animal reluctant to open its mouth for examination.

3-On external palpation the animal show the signs of pain & coughing.

4-Pyrexia or fever in case of infectious agent.

5-Regurgetation of food and water from the nostrils in sever cases.

5-Presence of mucpureulent nasal discharge.

6-Dyspnoea, dysphagia and dysphonia.

7-Extension of the head and neck.

8-Salivation or drooling of the saliva with frequent tentative jaw movements.

9-Enlargement of parotid and retropharyngeal lymph nodes is common.

10-In case of pharyngeal phlegmon there are:

A-Acute onset of fever (41-41.5C).

B-Rapid heart rate with dyspnoea and depression.

C-Death occurs within 36-48 hours after the first signs of illness.

D-Pronounced obstruction to swallowing and respiration.

E-Diffuse swelling of the area of throat and it can be diagnosed easily by endoscopies.

11-In case of: traumatic pharyngitis there are :

A-Internal examination of the pharynx we found hyperemic tissues with lymphoid hyperplasia.

B-Erosions covered by diphetheric membrane.

C-Laceration of the pharyngeal mucosa is visible.

D-Accumulation of the ruminal ingesta in the lacerated mucosa or in the diviticulae on both side of the glottis.

E-External palpation on the most proximal part of the neck reveals pain and detected the diverticulae that contain ruminal ingesta.

12-Acute pharyngitis may be subsided within 3-4 days while chronic form may persist for several days or weeks specially if there is laceration of the pharyngeal mucosa or persistent foreign body.

13-Swallowed food is regurgitated few minutes after swallowing.

Diagnosis:

1-History and clinical symptoms.

2-Internal examination by laryngoscope.

3-P.M lesions:

A-We found the necropsy restricted in the pharynx.

B-In case of phlegmon we found swelling with abscess formation and in cut section there is fetid odor and some gases usually escape.

4-Differential diagnosis:

Disease / Points	Pharyngitis	Pharyngeal obstruction	Pharyngeal paralysis
On set	Sudden	Slow, if there is no foreign body.	Slow
Cough	frequent	Continuous	- ve

Systemic disturbances	+ ve	- ve	- ve

Treatment : ***A) Hygienic treatment:***

1-Prevent irritation by gases or smokes.

2-Isolation of the diseased animal specially in case of infectious agents.

B) Supportive treatment:

Fluid therapy as dextrose or saline to compensate loss of natural feeding.

C) Medicated treatment:

1-Anti-microbial drugs to kill microorganisms:

A-Salphademidin sodium 331/3 % Inj. 1 ml kg B.wt I.V., I.M.

B-Salphathiazole: no gm orally then 60 gm after 12 hours and every 12 hours for 5 days.

D-Oxytetracycline 2-3 gm daily by mouth or injection for 5 days.

E-Ampicillin 2-3 gm daily for 5- days.

2-Analgesic and local anesthetics.

2-Pharyngeal Obstructions

Definition:- Pharyngeal obstruction means that decrease the caliber of the pharyngeal lumen that interfere with swallowing and respiration. Characterized clinically by dysphagia, dyspnoea and abnormal respiratory sound (strediros)

Etiology: Maybe due to:

A-Foreign bodies as bones, corn cabs, wire or metal.

B-Tissues swellings as in case of:

1-Retro pharyngeal lymphoadenopathy or lymphadenitis.

2-Goiter.

3-Abscesses due to TB. – Actinobacillosis – Bovine viral leucosis.

4- Fibrinous or mucoid polyps. These are usually pendulated and it may cause intermittent obstruction of the air and food intake.

Clinical Symptoms:-

1-Dysphagia and dyspnoea with frequent and explosive cough.

2-Regurgitation of food from the mouth.

3-Snorting or abnormal breathing sound.

4-Prolonged respiration with marked abdominal effort.

5-Emaciation in long period due to reluctant of food.

6-Rapture of the abscessed lymph node spontaneously or by falls using of stomach tube leads to meucupureulent nasal discharge or aspiration pneumonia that leads to death.

7-Change in phonation.

8-Laceration of the tongue.

Diagnosis:-

1-History and clinical symptoms.

2-Laryngoscope and radiography.

3-Differential diagnosis:

A- Pharyngeal paralysis

B-Esophageal obstruction

C- Pharyngritis

Treatment:-

1-Remove the cause and treatment of the infectious diseases if it is suspected.

2-Removed of the foreign bodies from the mouth.

3-Parental treatment of abscesses by penicillin gives good result.

4- Surgical interference has been highly successful in cases which caused by abscess in the retro-pharyngeal lymph node.

3-Pharyngeal Paralysis

Definition:- Pharyngeal paralysis is more common disease in farm animals, characterized by inability to swallow and absence of the signs of pain and respiratory obstruction.

Etiology:- ***A- Peripheral nerve injury due to*** :

1- Trauma to the throat region. 2- Guttural pouch infections in horses.

B- Secondary to specific diseases as in case of:

1- Botulism 2- Rabies and other encephalomyelitis.

Clinical Symptoms:- 1- Inability to swallowing.

2- Absence of the signs of pain and respiratory obstruction.

3- Cud- dropping and there is difficulty in controlling the regurgitated bolus which is often dropped from the mouth during rumination.

4- The animal is hungry, but during the attempts of prehension of food or water there are: - a- Dropping of food from the mouth.

b- Expulsion of food from the nostrils. c- Coughing [explosive cough].

5- Excessive salivation, and swallowing cannot be stimulated by external pressure on the pharynx.

6- There is rapid loss of condition and dehydration.

7- Usually there is no – systemic disturbance except if the cause is infectious as in case of botulism or rabies.

8- Aspiration pneumonia may occur due to dropping of the regurgitated food through the nostrils into the trachea and lung leading to aspiration

pneumonia [fatal] which characterized by loud gurgling sound of the lung by auscultation. 9- Roaring sound during breathing.

Diagnosis:-1- Clinical symptoms and History. 2- Endoscope.

Treatment:-1- Treatment is unlikely to have any effect in such condition.

2- Local applications of hot fomentation may be attempts.

3- Intravenous or stomach tube feeding may be tried to overcome the unthrift ness

4-Esophogitis

Definition:- Inflammation of the esophagus which clinically characterized by pain on swallowing and palpation, regurgitation of blood – stained slimy material and signs of spasm and obstruction initially.

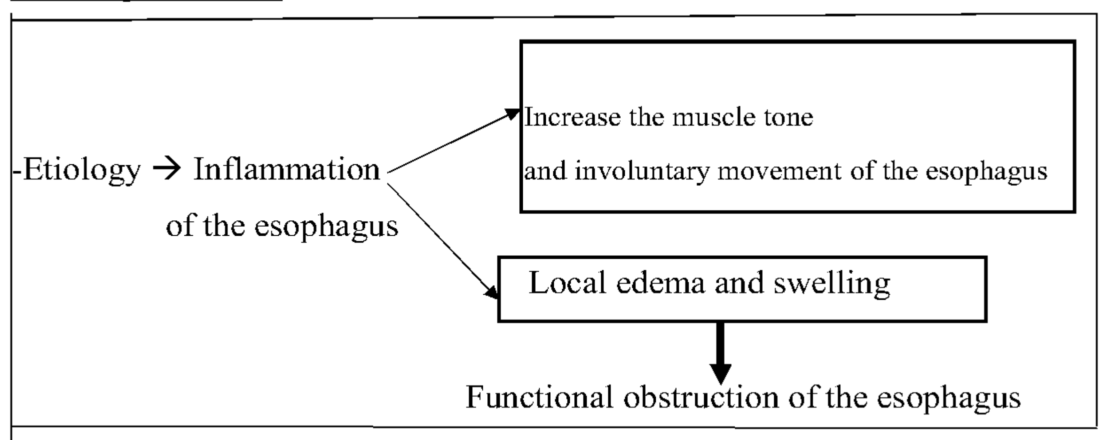
Etiology:-1- Primary esophogitis usually caused by ingestion of irritant material and it usually accompanied with stomatitis and pharyngitis.

2- Laceration of the mucosa of the esophagus by stomach tube or foreign body but it's not accompanied with lesions elsewhere.

3- Death of the hypoderma lineatum larvae in the mucosa of the esophagus may cause acute local inflammation and subsequent gangrene.

4-All causes of stomatitis [many specific diseases] usually associated by esophagitis but the other clinical signs of these diseases overshadows the signs of esophogitis.

Pathogenesis:-



Clinical Symptoms:-

1- In the acute stage: there is salivation and attempts of swallowing which cause sever pain.

2- In some cases: swallowing is impossible and attempts to do so are followed by → regurgitation, of food, coughing and pain.

3-Retching movements and vigorous contractions of the cervical and abdominal muscles.

3- The regurgitated food may contain blood or mucous.

4- If perforation of the esophagus occurs it loads to:

A-Local swelling and crepitating or fistula.

B-May leads to pleurisy and death.

5- Animals which recovered from esophagitis are commonly affected by chronic esophageal stenosis with distension above the stenosis.

Diagnosis:-

1- History and clinical symptoms

2- Differential diagnosis:

A-Pharyngeal paralysis:- There is less existence of pain on palpation.

B-Complete esophageal obstruction: Usually accompanied with tympany

C- Pharyngitis:
- →Swallowing may be normal
- →Coughing is frequent but esophagitis occurs commonly to getter with pharyngitis.

3- By endoscope to examine the esophageal mucosa.

4- By using of stomach tube to detect the location of the foreign bodies.

Treatment:-

1- Food should be withheld for 2-3 day and parental feeding is needed in this period.

2- Potential administration of anti-microbial agents [antibiotics or sulphonamide] to compete the secondary bacterial infection.

3- Reintroduction to feed should be careful and monitored and the feed should be moistening to avoid the possible accumulation of any food on the esophagus which may not be fully functional.

4- Anti – inflammatory and analgesic to relief the pain.

5-Esophageal Obstruction "Choke"

Definition:-* Means obstruction or occlusion of the esophagus which may be acute or chronic characterized clinically by: a-Inability to swallow.

B-Regurgitation of food and water from the mouth in cattle and nostrils

C-Continuous drooling of the saliva and bloat.

Etiology:-A-Obstruction may be intra luminal by ingested material.

B-Extra luminal by the pressure on the esophagus by the surrounding organs or tissues.

A- Ingested material :

1- Solid obstructions specially in cattle by peaches, apple turnips, potatoes

2- Broken fragments of naso-gastric tube and left in place for over period of time.

3- Foreign bodies as woods, wire…… Etc.

4- Accidental swallowing of the stomach tube.

d- Feeding on large quantities of dry food or grain can cause obstruction.

B- Pressure by surrounding organs or tissues or abnormality of the local tissues of the esophagus:

1- TB.or neoplastic lymph nodes in the mediastinum or at the base of the lung.

2- Persistent right aortic arch. [Congenital – anomalies].

3- Carcinoma of stomach causing abstraction of the cardio.

4- Esophageal hiatus hernia in cattle.

5- Granulation tissues as a result of previous laceration of the esophagus

7- Esophageal phytobezoar. 8- Esophageal mucasal granuloma.

9- Cervical or mediastional abscesses.

10-Traumatic rupture of the esophagus as a result of kick or using of nasogastric tube.

11-Congenital hypertrophy of the esophageal musculature.

Pathogenesis:-

***1- In acute obstruction there is*:** initial spasm. At the site of obstruction for forceful, painful peristalsis and swallowing movement.

2- Complications of the esophageal obstruction may be :

a- laceration and rupture of the esophagus.

B-Esophagitis, stenosis and development of esophageal diverticulum.

c- Necrosis of the site of obstruction.

d- Rapid development of bloat.

3- If the site of obstruction:-in the cervical part of the esophagus it can be palpated at the left jugular furrow and the prognosis in such condition is more good than that if the site of obstruction in the thoracic part of the esophagus.

4- Esophageal obstruction may lead to mega-esophagus in which the esophagus is dysfunctional, dilated and filled with saliva, food and water which leading to regurgitation of food and water which may lead to fatal Aspiration pneumonia.

5-" Esophageal diverticulums": which described as sac (circumscribed sac)

of the mucosa protruding through the muscular layer of the esophagus which may be due to.

A- Excessive intraluminal pressure from impacted food or foreign body in the esophagus.

B-Fluctuation in the esophageal pressure due to obstruction.

Clinical Symptoms:-

A- Acute esophageal obstruction (Choke):

1-The obstrcution usually in the cerival part of the esophagus just above the lanynx or at the thoracic inlet, Also it may accur at the base of the heart or the cardia. [usually sporadic cases].

2-The animal suddenly stop eating and shows anxiety and restlessness.

3-Forceful attempts to swallowing.

4-There is → regurgitation, salivation, coughing and continuous chewing movements.

5-If the obstruction is complete ruminal tempany occurs rapidly and the animal become very difficult to be handaled.

6-Ruminal movement is continuous forceful with systolic murmur audible on auscultation of the heart.

7-The acute signs, other than bloat usually disappeared within few hours this due to relaxation of the initial esophageal spasm and may or may not a combined by onward passage of the obstruction.

8-The obstructions may be pass spontaneously or may be persiste for several days or weeks with continued bloat and in such conditions ther are

: -In ability to swallow. - Salivation and continued bloat.

- Passage of stomach tube is impossible.
- Pressure necrosis on the mucosa at the site of obstruction which may lead to perferation of the esophagus and consequently may lead to fatal pleurisy or subsequent stenosis due to fibrous tissue formation.

9- In case of incomplete esophageal obstruction the water only can pass .

10-If the obstuction due to feed it can occurs at any level of the esophagus

11-External paplation of the cervical esophagus may reveal a firm cylindrical swelling a long the course of the neck on the left side when the esophagus is obstructed with food or foreign body.

12-Etension of the head and neck with forceful attempts for swallowing with salvation and regurgetation of food and water .

13-Death usually due to : aspiration pneumonia or when the obstruction persisite from dehydration.

B- Chronic obstruction:-

1-Absence of the signs of acute form.

2-Chronic bloat.

3-Rumen may continue to move .

4-The swallowing movement is normal until the bolus of the swallwing food reach the site of obstruction.

5-The swallowed material either pass slowly through the stenotic area or accumulated over it and then regurgetated.

6-Stomach tube can pass.

7-There is no attempts made to eat solid food but fluid may be taken and swallowed easly.

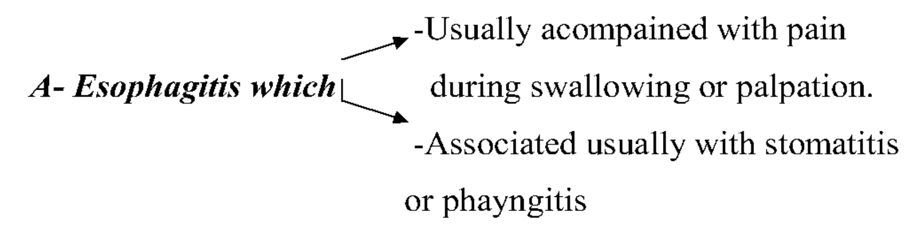
8-When there is esophageal paralysis as in megaesophagus regurgetation may not occurs and Aaspiration pneumonia may occurs leading to death.

Diagnosis:-

1-History and clinical symptoms.

2-By using of stomach tube and endoscope or contraste radiography to detecte the site and nature of the obstruction.

3-Differential diagnosis :



B- Diseases characterized by dysphagia or tempany or that leading to reguragetation of food.

NB. Stomach tube can be used to detecte the site of the obstruction either in the cervical or in the thoracic part of the esophagus as well as to detecte either the obstruction is incomplete (can be pass)or complete (cannot be pass)

Treatment:- "Prevent food intak with excessive water uspply"

1- Sedation of the animal to help in relaxing the esophegeal spasm by using of : -Cholorprmazin Hel 50-100 mg/kg.-Chloral hydrote. 3-4

gm/50kg Bwt. – Neurazin – Rempon – combelen.- Atropin Sulphate 16-32 mg S/c.

2- Pushing the obstruction by using of the stomach tube inwarde and toword the stomach but carful must be taken to avoid damage to esophnageal

3- If the foreign body in the cervical part of the esophagus it can be reached by mucosa.passing the hand in the mouth [using gage] and press the foreing body from external by assistant the movement of the foreign body toward the mouth.

4- Trocar and canula to releife the tempany.

5- Long piece of wire can be bent into a loop may be passed over the object and an attempts to pull it into the pharynx(not perifearable).

6- Carful lavage of the obstructed esophagus by worm salin or water to remove the accummulated food above the site of obstruction. By using stomach tube pump the water or salin in the esophagus and syphen it by gravity and lowring the head of the animal this process is repeated until the salin or water come clear but care must be taken to avoid overflow of the esophagus and fatal aspiration pneumonia [proper amount of water or salin per time about 0.5- 1 liter].

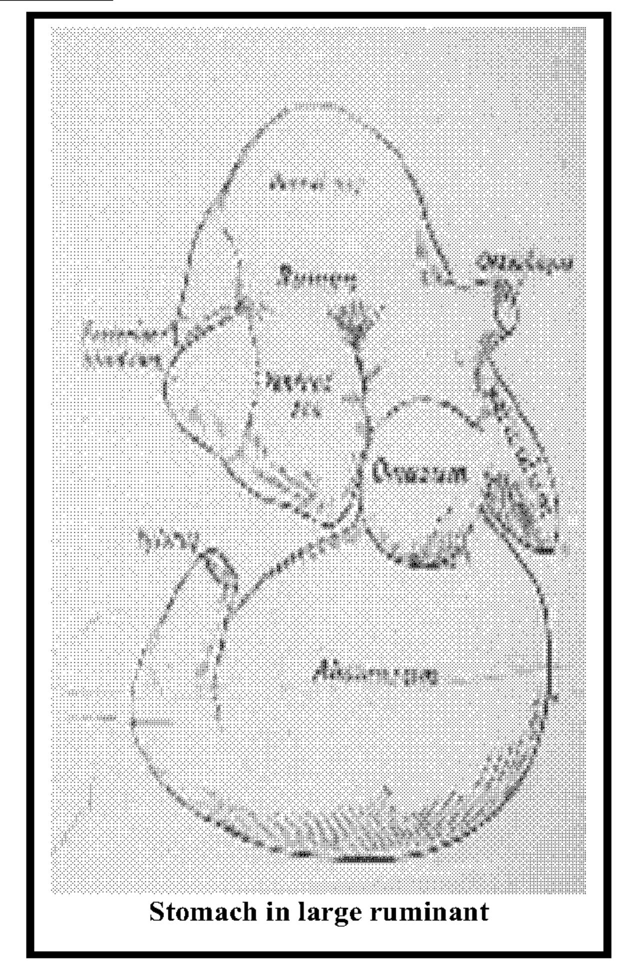
7- Surgical interference by the esophagotomy but it's complications may leads to narrowing or stenosis of the esophageal lumen due to scare tissues formation after the surgical operation.

8- Parenteral feeding is required at the period of treatment to avoid dehydration and unthirftness.

C-Diseases Of The Stomach

1-Introduction

(I)- Anatomical considerations :


Stomach in large ruminant

*The stomach in the ruminant animals consists of four compartments
[rumen- reticulum - omasum – abomasum]
and it's occupies about 75% of the abdominal cavity.

A-Rumen (Booming or gurgling sound)

1-It's the largest compartment, it's form about 70-80% of total size of the stomach.

2-It occupies the left side of the abdomen.

3-It can be examined at the left flank region. (hunger fossa).

4-It's capacity about 60-80 gallons of water.

5-Consistes of dorsal ruminal sac (DRS) and ventral ruminal sac (VRS).

B- Reticulum : (swishing sound).

1-It follows the rumen and forms about 7-10% of the total size of the stomach.

2-It lies anterier to the xiphoid cartilage

opposite to 7^{th} left inter costal space

[6-8^{th} left ribs]can be examined on the left side of the abdomen.

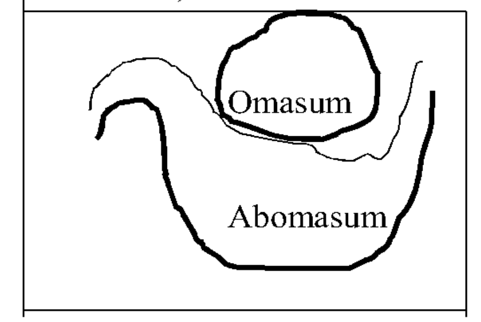
C-Omasum:

1-It follows the reticulum and forms about 5% of the total size of the stomach.

2-It lies right to the medium plain opposite to the 7-8^{th} ribs on the right side. Over the abomasum.

3-It's left side (surface) is contact with the abomasum and rumen while it's right surface is contact with the diaphragm and liver and abdominal wall, it has dorsolateral fold. [it cannot be examined clinically].

D- Aboma sum : (High pitch tinkling or metalic sound).



1-It's the true part of the stomach resembling to the stomach of the monogastric animals [forms about 8-10% of the stomach].

2-It lies on the abdominal floor on the right side.

3-The anterior wide end is related

the reticulum on the xiphoid cartilage while the posterior narrow end is related to py lorus opposite to the 9th or 10th rib on the night side.

4-It's greater curvature is contact with the abdominal wall while the lesser curvature caries the omasum.

(II)- Functions of the stomach:-

1- Bacterial digestion and fermentation :

By the action of the ruminal microflora and microfauna on the ingested material to produce volatil fatty acids formed [75% acetic acid , 15% propionic and 10% butyric acid], carbone dioxide, methan and other derivatives of amino acids deamination such as amins and histamine.

2- Pahysical maceration :

By movement of the of the rumen and reticulum – These two main functions are interdependant on each other, so that ruminal movement is used as an index of the digestive function.

(III)- The layers of food content in rumen:-

1-Free gas layer(FGL).

2-Fluid, food suspanded particles and air bubles layer.

3-Heavy and coarse particle layer which is the most down layer.

FGL
Mixed
Heavy

(VI)-Types of ruminal movement:-

A- Eructation movement :

- It occurs by the contraction of the dorsal sac of the rumen (air containing

layer of the rumen)in the form of secondary ruminal movement.

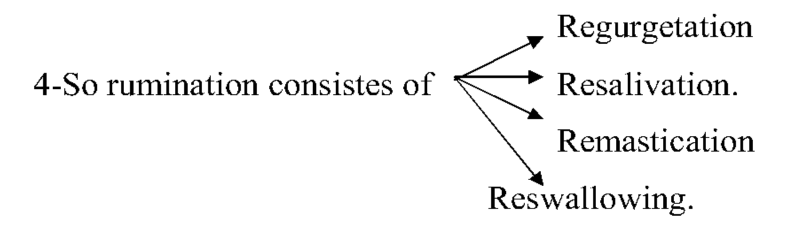
B- Rumination movement :

1-It occurs by deep inspiratory effort to creat negative pressure in the thorax followed by escaping of the reticular fluid to the cardia then to the esophagus which stimulates the antiperstaletic movement of the esophagus.

2-The antiperstaletic movement of the esophagus push the fluid and it's containing food material toward the pharynx then to the mouth then resalivation and remastication then reswallowing is occurred.

3-Rumination is stimulated by the presence of coars fibres found in the rumen and reticulum, so the roughage part of the ration is very important for enhancing the rumination.

4-So rumination consistes of



C- Mixing movement :

1-It begins by double movement or contractions of the reticulum followed by contraction of the anterior dorsal ruminal sac .These movements allowed the fluid in the reticulum return back to the rumen.

2-The dorsal sac contraction make a sort [type] of rotation in the rumen, this rotation is completed by contraction of the venteral rumenal sac .

3-The end result of all these contractions is the maceration and mixing of the food particles which found in the rumen.

1-Simple Indigestion

Definition:- It's a case of indigestion characterized by inappetance to anorexia, decreased ruminal movements and abnormal feces which may be scanty, voluminous or diarrheic. That disease usually associated with dietary abnormality.

Etiology:- 1- The disease is more common in dairy cattle and stall – fed beef cattle due to variability in quality and the large amount of food consumed, and not – common in pastured beef cattle , that due to they are less heavily fed.

2- Feeding on indigestible roughage with low protein intake.

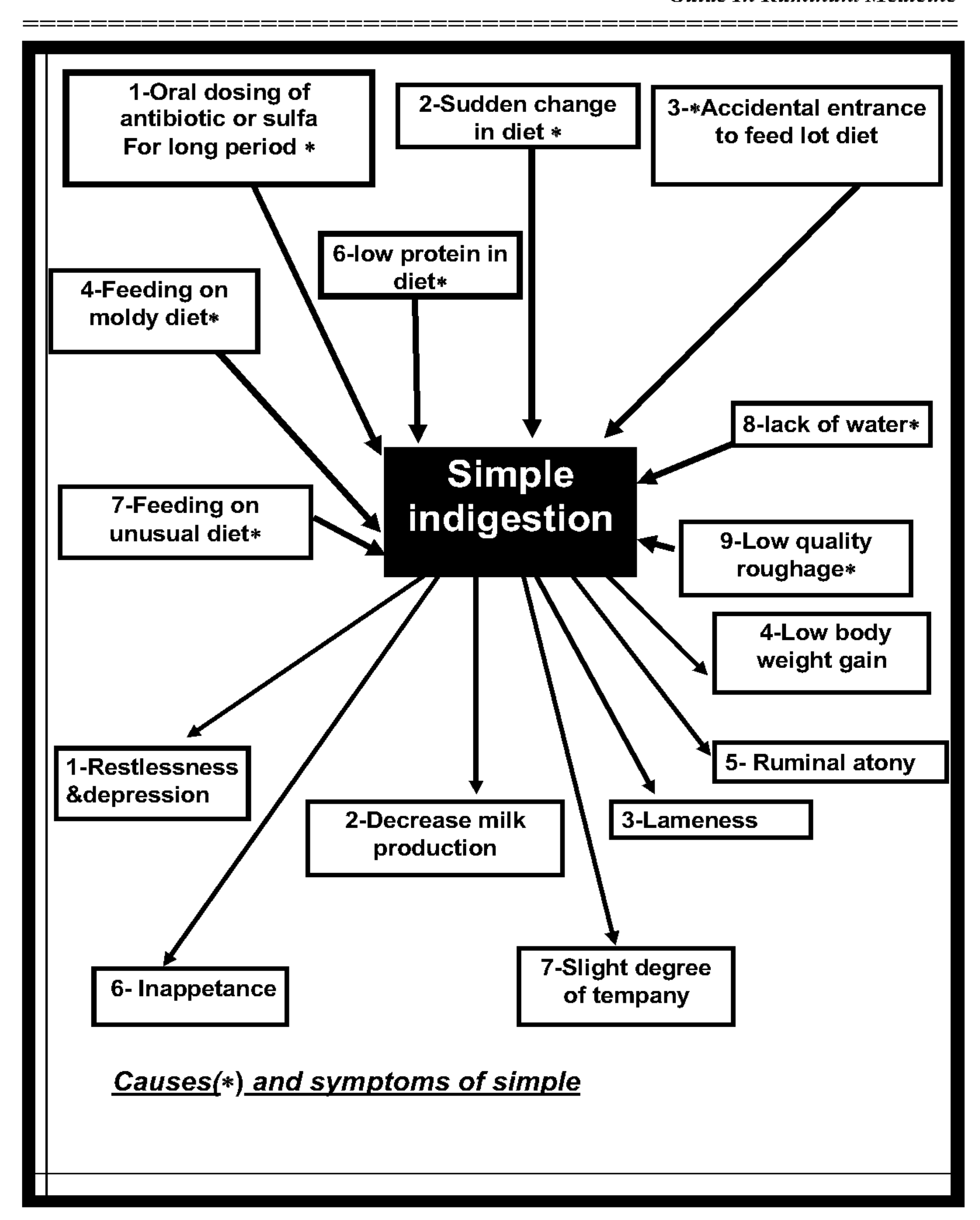
3- Moldy, over heated, frosted [freeze] feed and moderate excess of grain and concentrates i.e. over feeding with grain.

4- Gross over feeding usually occurs when cattle gain accidental access to large quantities of grain or are suddenly introduced to high grain diet in feedlots.

5- Sudden change to a new source of food.

6- limitation of the available drinking water specially in dry seasons.

7- Prolonged or heavy oral dosing of sulphonamide or antibiotics may lead to indigestion due to inhibition or inactivation of the normal ruminal microflora. That leads to decrease the amount of fatty acid and consequently decrease the milk production.



Causes(*) and symptoms of simple

Pathogenesis:-

1- Over feeding on grain leading to increase the ruminal acidity which has markedly affect on the motility on the rumen that leads to ruminal atony or even complete ruminal stasis if the ruminal PH less than 4-5.

2- Over feeding on high protein diet as legumes or urea also leads to depressed ruminal motility due to the sharp degree of alkalinity which resulted from overfeeding of this type of food.

3- The simple accumulation of the indigestible feed in the rumen may lead to depression of the ruminal movements.

4- Putrefaction of protein in the rumen leads to production of amides and amines as histamine that leading to ruminal atony and lameness due to arthritis with unknown interpretation.

***<u>* Experimentally</u>*:** *We found that administration of histamine intravenously lead to ruminal atony and the effect is depressed after administration of anti histaminic drugs, so that is the main cause of ruminal atony in the allergic conditions.

5- Ruminal atony leads to →

- Low production of the volatile fatty acids this reflected on low milk production
- Decrease the food intake [anorexia] that due to the ruminal contractions play the same role of the hunger contraction of the stomach in the monogastric animals.

Clinical Symptoms:-

1- Some degree of inappetence to anorexia.

2- Slight decrease in the milk production.

3- Ruminal atony or stasis by auscultation. [N= 2-5 movements/2min.].

4- Mild degree of depression and dullness but the posture of the animal is not affected.

5- Rumen may be larger than normal specially if there is history of feeding on unlimited amounts of palatable food.

6- Some cases there is slight degree of tempany specially when there is feeding on damage, frozen or moldy food but the usually finding in the rumen is [firm, doughy on palpation without obvious distension].

7- The feces is reduced in quantity and more drier than normal but after 24 – 48 hours it become diarrheic, voluminous and /or scanty.

8- There is no systemic reaction and the respiratory rate, temperature and pulse rates are within normal. And there is no – pain in deep palpation of the abdomen.

9- Most cases are recovered spontaneously or with simple treatments within 48 hours.

10- Low turned over rate [conversation rate] with loss of weight gain.

Diagnosis:

(I)- Case history and clinical symptoms.

(II)- Laboratory diagnosis:

A-Detection of the ketone bodies in the urine by sodium nitroprosside

test to differentiate with ketosis (-ve Rothar's test).

B-Detection of the ruminal activity of microflora …. Etc.. by:-

1- Cellulose digestion test [time more than 30 hours while the normal time is N= 24-30hours].

2- Sedimentation–flotation test [time more than 9minutes while the normal time is N=3-9 minutes]

3- Microscopical examination of microflora [less than 5 microflora /power field while the normal is N=5-7 active motile microflora per power field].

4- Examination of fungi in the ruminal juice [rhisobase–aspsedia …etc.)

5- Detection of the PH. Of ruminal juice [N= 6.2 -7.2].

(III)- Differential diagnosis: with diseases causes primary ruminal atony as diseases of rumen and abomasum or reticulum. And other diseases cause secondary ruminal atony as the following:-

1- Ketosis there are: * There is ketonuria (by Rother's test).
* The rumen is weaker than normal movements.
* The milk production is reduced over few days.

2- Traumatic reticuloperiotenitis there are:

*Sudden onset of anorexia, agalactia.
*Mild fever & tempany
*Painful grunt on palpation of the xiphoid sternum [positive pain test].
*The rumen is static and decreased in size.

3- Carbohydrate engorgement (Impaction)there are :

*Ruminal stasis with fluid splashing sounds

*Dehydration, tachycardia and depression.

*Staggering and recumbency.

* Ruminal PH. Lower than 6 [common less than 5]

4- Left sided displacement of the abomasums [L. D. A] There are.

* Ketonuria & small sized rumen in examination.

*Occurs usually within few days after parturition

*Ping sound on percussion in the lower left flank region (tinkling sound)

*The strength of the ruminal contraction is usually reduced.

5- Vagal indigestion there is:

*Gradual distention of the abdomen due to distension of the rumen over a period of several days.

*Progressive dehydration and scanty feces.

*Hypermotality of the rumen initially.

*Recurrent frothy tempany followed by ruminal atony.

6- Abomasal dilatation there is:

* Most common 2-4 weeks postparturent [in dairy cows].

*Presence of pinging sound over the right flank region.

*Distended viscous is palpable per-rectum in the lower right quarter.

*Inappetance reduced feces, ruminal atony and reduced milk production.

7- Hypocalcaemia there are: - Ruminal atony and anorexia in early stage.

*Recovery after treatment with calcium borogluconate.

Treatment:- Rational treatment is often difficult because of lack of knowledge of etiology, so the most part of treatment is symptomatic.

Consisting primarily of the rumenotonic drugs.

(I)- Hygienic treatment:-

1- Allow the animal to adequate water supply.

2-Examinatio of the food materials thoroughly to exclude moulds or any other decomposing substances.

3-Correction of the amount of carbohydrate or the nitrogenous compounds of the ration as urea, grains or legumes.

1- Withholding the diet at least 12-24 hours(fasting)

(II)- Medicated treatment:-

1-Rumenatoric or ruminal tonic:

A-Tarter emetics 10-12 gm. orally in diluted form to avoid the irritant effect of the drug which may lead to chemical reticulitis.

B- Parasympathetic stimulants: (parasympathomimetic): As,

* Carbamylcholine chloride- physostagmine 2-5 mg/100 kg and Neostagmin [most effective and given 2-5 mg /45 kg Bwt).

C-Carbamyl choline chloride act mainly on the muscular contractility, so it leads to uncoordination and functionless movements and it is contra indicated in late pregnancy & peritonitis.

N. B →

The normal flow of rumen contents from the reticulorumen. to the abomasum is the result of synchronized contractions and relaxation of the various parts of the forestomachs, orifices and abomasum which occurring simultaneously, so one of the most limitation of the injection of

parasympathomimetic preparations when it is used as rumenatorics is that they don't provide these synchronized movements and therefore there are unnecessary movements which may lead to abortion in the pregnant animals as in case of using of R/ Carbacol 1ml /100 kg. s/c.(over doses lead to ruminal stasis but small doses and repeated within short intervals can gives good result). Also it is usually associated with excessive salvation & tremors (parasympathetic stimulant)

2- Purgative :-

1-Mineral oil as Paraffin oil it acts as mechanical purgative which envelop the food particles and lubricate it to be easily passed through GIT.

2- Saline purgative such as → Epsom salt (1-2lb orally)

It acts by increasing the water content of the intestine and consequently stimulate the peristaltic movement of the intestine via the afferent and efferent fibers of the vagus nerve.

3- Stomachic:-

1-It acts by increasing the appetite via the hunger contraction, so that stimulate the contractility of the compound stomach.

2- We can use strychnine preparation up to 65 mg as a single dose (R/ Stomavatic, Vapcodigest –Stomatone – Stomavetic Digestine……etc.

4- Gastric stimulants:-

R/ - Ammonium Carbonate 16gm.

- Oil of turpentine 30cc.
- Linseed oil or water up to 1 liter.

3 times within interval 12 hours if necessary.

5- Adjustment of the ruminal PH:-

1- In case of increased acidity: We can use substances of alkaline radicals as

→ Magnesium hydroxide 500 gm dissolved in water.

→ Sodium bicarbonate 200-500gm orally or I.V. 2.5 -5%

2- In case of increased alkalinity: We can we substances of acidic radicals as :

- Vinegar 50- 100 ml or diluted Hcl.
- Acetic acid 5% (2ml/kg Bwt.).

6-Anti–histaminic:

To correct the harmful effect of the histamine (R/Avil 3 ampules i.v. or i.m).

7- Ruminal Juice Transplantation:

1- We can reconstitute the ruminal flora specially when there is change in the ruminal PH. of the diseased animal.

2- We can obtain ruminal microflora from the slaughtering house or from the healthy living animals [1-2 liters].

3-Ruminal Impaction

Synonymes :-

1- Grain poisoning. 2- Grain engorgement or overload.

3- Acute ruminal acidosis. 4- Founder.

5- Acute indigestion. 6- Lactic acidosis.

7- Carbohydrate engorgement.

Definition:- It's acute disease occurs due to ingestion of large amounts of highly fermentable carbohydrate rich diets which leads to excessive production of lactic acid in the rumen. Clinically characterized by sever toxemia, dehydration, ruminal stasis, anorexia, weakness, recumbancy and high mortality rate.

Etiology:-1-Mainly the sudden ingestion of toxic doses of carbohydrate rich diet such as grains.

2-Less common causes including engorgement of apples, bread, or sugar beat.

3-Ingestion of large amount of diets containing non–protein nitrogenous material as urea [normal rate of urea in the ration must not be more than 3%.

Epidemiology:-

A- Predisposing factors :

1- All types of ruminant including cattle, sheep, goat and deer specially feed lot cattle that fed on carbohydrate – rich fed [grain] are more susceptible.

2- Accidental consumption of toxic level of grains by cattle which gain sudden access to large quantities of stored food or grains.

3- Providing of unaccustomed quantities of the grains to the animal.

4-Feeding of green unripe corn.

5- Miscalculating of the ration constituents.

6-Sudden change in the diet from green or roughage ration to complete dry concentrated ration that leads to disturbances or change in the microbial population in the rumen.

7- Feeding on feed containing mold which help the growth of fungi in the rumen [Absedia – Rbisopus & Mucore].

8- Feeding on large amount of ground finely grain.

9- Long period of hungry about 12-24 hours followed by feeding of an unlimited supply of feed will often result in sever cases of grain overload (Impaction).

B- Morbidity and mortality :

1- Morbidity rate up to 10-50% of the herd which kept on grain farm and feed lots.

2- Mortality rate: up to 90% of untreated cases while in treated cases mortality may reach up to 30-40%.

C- Types and toxic doses of feed:

1- Wheat, barely and corn grains are considered to be the most toxic when ingested in large quantities while oats and sorghum are less toxic.

2- All of the grain are considered much more toxic when ground finely or

even rushed or just cracked.

** Experimental feeding on unprocessed barely to cattle did not result in rumenitis while feeding on finally ground barely resulting in ruminal lesions.

3- The toxic dose of grain that able to produce the disease mainly depend upon

a- Types of grain. b- Nature of the microflora.

c- Previous experience of the animal to such types of grain.

d- Nutritional status of the animal and it's bodily condition.

4- Dairy cattle accustomed to heavy grain diets may consume 15-20 kg of grain with develop of only moderate illness while beef or feedlot cattle may become acutely ill and die after eating of 10 kg of grain.

5- The toxic dose of the grains in cattle which my produce lactic acidosis is 25-62 gm.grains/kg Bwt. of ground cereal grain or corn may produced sever acidosis.

Pathogenesis:-

1- Ingestion of large quantities of highly fermentable carbohydrate rich diet

↓ ← Within 2-6 hours after ingestion.

-Marked change in microbial population of the rumen which are:-A-Increase the number of Gram positive acidophilus bacteria as streptococcus bovis.

B-Decrease the numbers of Gram Negative cellulytic microorganism.

2- Excessive number of Gram + ve strep. Bovis leads to utilization of carbohydrate and production of large amount of lactic acid.

3- Presence of sufficient amount of carbohydrate [toxic or lethal dose in the rumen lead to flourishing of the strep. Bovis resulting in :-

A-Excessive production of lactic acid in the rumen.

B-Lowering the ruminal PH. until 4-5

C-Destruction of the gram negative cellulytic bacteria, protozoa and microflora.

4- Lactic acid is stronger than volatile fatty acids (which produced normally in the rumen) by ten times, so it's absorbed rapidly and causing acidity or academia.

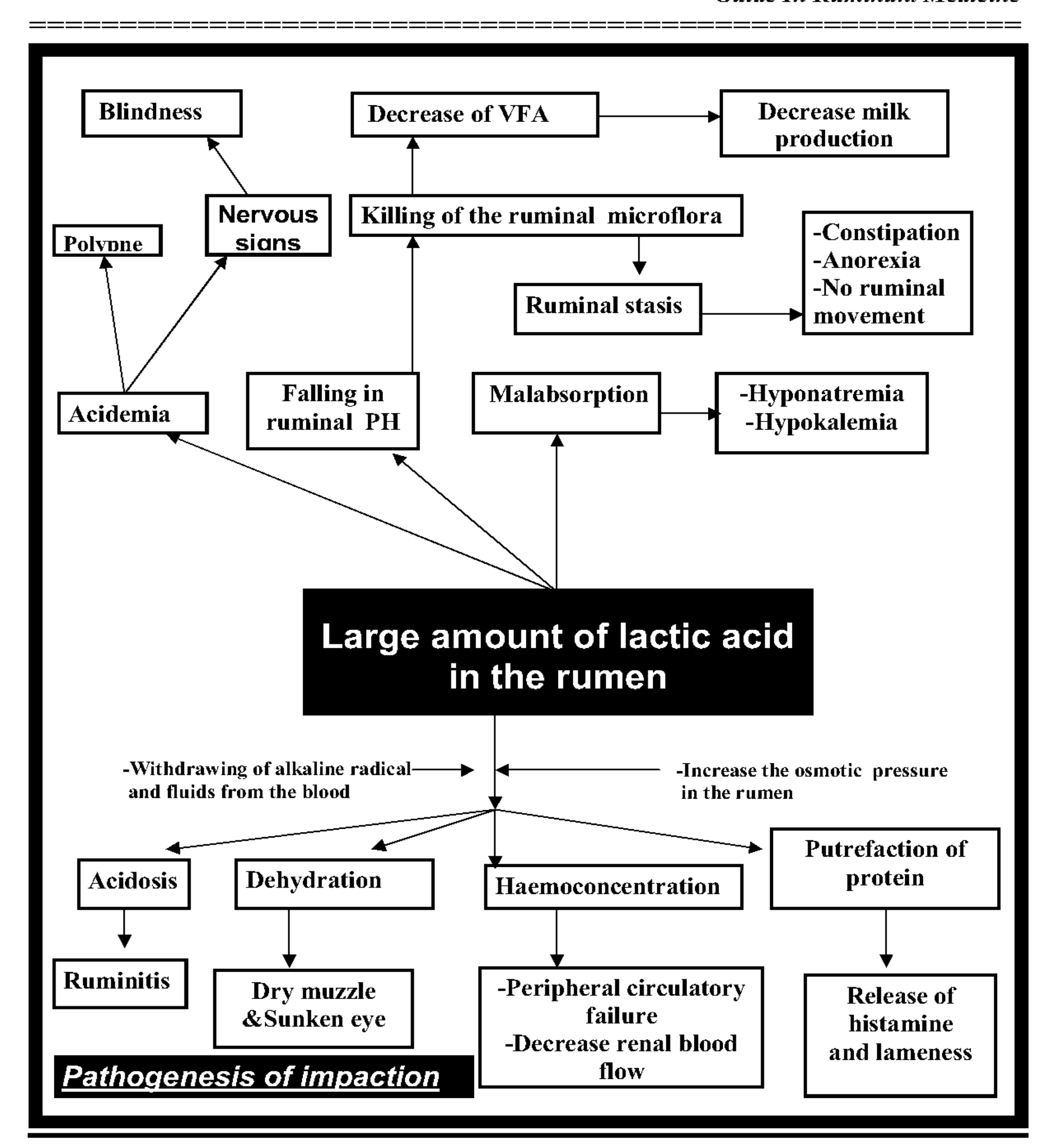
5- Ruminal stasis is suggested to that lactic acid may reach the duodenum and exerting a reflex inhibitory action of the rumen.

6- Acidosis may be preceded by alkalosis due to some of absorbed lactate may converted into bicarbonate in the first few hours of absorption of lactic acid.

7- Diarrhea may occur due to reduction in the net absorption of water form the colon. (Good prognosis).

8- Follow the next diagram of pathogenesis:

9- When the animal fed on high amount of protein nitrogenous compound (urea) it leads to production of high amount of ammonia which cause increase the ruminal PH toward the alkaline side this also will lead to suppression of the normal microflora and the degree of production of the volatile fatty acids.



Pathogenesis of impaction

Clinical Symptoms:-

1-The speed of onset of the disease varies with the nature and amount of the diet [the disease being faster with ground food than whole grain food].
2-Abdominal pain which evidenced by kicking at the belly.
3-In mild form the cattle are anorexic, but still fairly bright and alert with soft feces or diarrhea is common.
4-Constipation followed by diarrhea.
5-In sever case the animal become recumbent within 24-48 hours.
6-The affected animal become off food [complete loss of appetite], and depressed.
7-Teeth grinding specially if there is sever degree of the abdominal pain.
8-Temperature usually normal or sub- normal with cold extremities.
9-Heart rate is increased and continues to increase with the severity of acidosis and circulatory failure.

N. B→

In general the animal with heart rate below 100/min. are considered much better than those with rate up to 120-140/min. ,so the heart rate is a good prognostic aid in this case [N= 55-80/min].

10- The respiration is usually shallow rapid up to 69-90/min. [N= 10-30/ min].
11- Diarrhea [profuse and the feces are light colored with an obvious sweet – sour odor and may contain excessive quantities of grains or undigested food particles).

12- Sever dehydration [degree may be loss of 4-6% or 10- 12% of body weigh].

13- Partial or complete ruminal stasis by auscultation [N= 2-5 movements /2min Booming sound].

14- On palpation of the left Para lumber fossa we detect doughy and firm palpation with degree or resilient sound due to slight degree of tempany.

15- Gurgling or splashing sound by auscultation due to accumulation of large amount of water and other body fluids in the rumen.

16- Lameness and severely affected animals reluctant to move.

17- Anuria is common and diuresis is occurred after treatment.

18- The recumbent animal may take position similar to that of parturient paresis [the head is turned on the shoulder toward the flank region] and sluggish of response [but in hypocalcaemia the feces is firm &dry and response to calcium therapy].

19- Death occurred within 24- 72 hours.

20- In sever persistent case of pregnant animals abortion may occur within 10 days to 2 weeks later.

21- Distention of the left Para lumber fossa with lowering of the head.

Diagnosis:-

(I)-History and clinical symptoms are characteristic.

(II)-Laboratory diagnosis:

A- Examination of ruminal Juice:

1- Cellulose digestion test is more than 30 hours [N= 24-30hr].

2- Sedimentation floatation test is more than 9 min [N= 3-9min].

3- PH: Less than 6 [N= 6.2- 7.2 average 6.8].

4- Color: May be darkening black. Or blackish green.

5- odor: rancid or offensive odor.

6- Microscopical examination:

-Low number of protozoa less than 5/power field [N= 5-7/power field].

-Presence of Gram positive streptococcus.

-Presence of fungus [Rhisopus – Mucore and Absidia].

B- Blood examination :

1- Increase of PCV. up to 50-60% [N= 30-32%] due to dehydration.

2- Low level of bicarbonate due to acidosis and acidemia.

3- High level of inorganic phosphate and lactate.

4-Mild degree of hypocalcaemia 6-8mg%[N=8-12mg%] due to malabsorption.

5-PH.of the blood is markedly decreased due to academia

C- Acidic and concentrated urine:-

D- PM. lesions :

1- In sever acute cases the ruminal and reticular contents are thin and have a typical odor suggestive of fermentation.

2- Ruminitis, perforation of ruminal wall may occur.

3- Peritonitis and liver abscess if the ruminal wall is perforated.

4- Abomasitis and enteritis may occur.

5- The abomasums contains large amount of grains.

Treatment:- The main principles of treatment are :

(1) Evacuation of the rumen.

(2) Correction of the ruminal and systemic acidosis and prevention of the production of further amounts of lactic acid.

(3) Restoring the fluid and electrolyte losses and maintain the circulating blood volumes.

(4) Hygienic and supportive treatment.

(5)Ancillary and supportive treatment (Ruminal tonics, Antihistaminic..etc.)

(1) Evacuation Of The Rumen :

A-Oil purgative [1-2 liters of mineral oil or vegetable oil as paraffin oil].

B- Surgical rumenotomy.

C- Pumping of 10-15 liters of warm water by stomach tube in the rumen and siphon it by gravity [it's repeated until the distension of the left Para lumber fossa is decreased].

(2) Correction Of Ruminal & Systemic PH.:

A-Intravenous administration of 2-5% solution of sodium bicarbonate. (5 liters / 450kg Bwt.).

B-Orally: → Sodium bicarbonate 200- 500gm.

→ Magnesium hydroxide or magnesium bicarbonate (250-400gm) followed by 120 gm after 12 hours.

And every 12 hours until recovery.

→ Lime stone (250-1100gm) followed by 120 gm after 12hours.

And every 12 hours until recovery.

→ The oral drugs are dissolved in 10 liters of warm water and pumped in the rumen followed by massaging of the of the rumen to promote the mixing movement.

(3) Correction Of Dehydration:

A- 0.9%Saline solution or 2-5% sodium bicarbonate 1-2 liters intravenous in case of academia.

B- Ringers lactate in case of alkalosis. 1-2 liters intravenous.

(4) Hygienic Treatment :

A-Avoiding the excess of grain.

B- Providing palatable hay or roughage (not less than 10%).

C- Massaging of the rumen by the hand.

D- Exercise of the animal to promote the movement of the ingesta in the digestive tract.

E-Withholding of water and food for 12-24 hours (fasting).

(5) Ancillary And Supportive Treatment :

A-Anti – histaminic for competing of laminitis.

[Avil – Tavagyl – Allecure – Vetibenzamine].

B-Calcium borogluconate 2% 1-2 liters I.v. as tonics and to promote the ruminal movement

C-Corticosteroids for shock therapy [dexamethasone].

D-Brewer's yeast (thiamin) for promoting the metabolism of lactic acid.

E-Parasympathomimetic to stimulate the gut motility as carbamyle choline chloride, physostagmine or Neostagmine [R/ carbacole 1ml /100 kg].

F-Stomachic for promoting the rumen motility after evacuation of the rumen:

R/ Stomaton – Bykahepar - Vapocodigest … etc.

G-Oral administration of antibiotics [penicillin or oxytetracyclin.] to control growth of unnecessary bacteria.

NB: Signs Of The Response Of The Treatment Are :-

*Lowering of heart rate to normal rate.

*Raising of the body temperature.

*Returned ruminal movement to the normal.

*Passage of large amount of feces.

*Normal appetite and responsiveness of the animal.

Prevention:-

1-Gradual changing of the ration eg. begin with 25% of grains plus 75% roughage [this ratio is gradually changed over one month to be 75% grains plus 25% roughage].

2-Addition of grounded lime stone and sodium bicarbonate equal amounts up to 6% of the ration.

3-Addition of gram positive antibiotic

4-Tempany or Bloat

"As mentioned in part (I) sheep & goat medicine"

5-Traumatic Reticuloperitonitis

Definition:-Traumatic reticuloperitonitis is an acute diffuse or local or chronic inflammation of the reticulum and peritoneum characterized by sudden anorexia and fall in milk yield, ruminal stasis and local pain in the abdomen and recurrent tempany.

Etiology:-1-Most cases are caused by ingestion of foreign body in the prepared feed.

2-Fencing wire which has passed through the food or forage harvester is the commonest cause of injury.

Occurrence:- 1-Cattle and buffalo are the most common susceptible animal because they use their tongue in prehension of the food.

2-Sheep and goats are rare to be diseased due to they are use their lips in prehension of the food and have selective ability to avoid ingestion of foreign objects.

3-It occurs in camel also [rare].

4-The disease is of great economic important because it's highly fatal disease specially in cute diffuse form of peritonitis.

5-Some of cases go unrecognized and make spontaneous recoveries.

Pathogenesis:-

(1) Lack of oral discrimination of cattle leads to ingestion of foreign body which can be rejected by other species as sheep and goat.

(2) The ingested foreign body may be lodged in the upper esophagus. Causing choke [esophageal obstruction]or in the esophageal groove causing regurgitation, but in the most cases the ingested foreign body pass to the reticulum.

(3) The foreign body by the vigorous contraction of the reticulum it may be lodged in the wall of the reticulum and give no harm or detectable illness.

(4) If the foreign body penetrates the wall of the reticulum it leads to many pathological problems.

(5) Penetration commonly occurs at the lower part of the anterior wall of the reticulum or may be occurs medially toward the liver or laterally toward the spleen.

(6) The foreign body may remain lying free in the reticulum but may leads to necrosis of the wall of the reticulum.

(7) Perforation of the wall of the reticulum leads to peritonitis which causes ruminal atony and abdominal pain and may lead to other complications as:

A- Traumatic pericarditis. B-Liver, spleenic or diaphragmatic abscess

C- Vagus indigestion. D- Diaphragmatic hernia [traumatic].

E- Rupture of gastroepiploic artery leading to sudden death due to internal

hemorrhage.

F-Arthritis, nephritis, endocarditis, pleurisy or pneumonia

G-Congestive heart failure and death

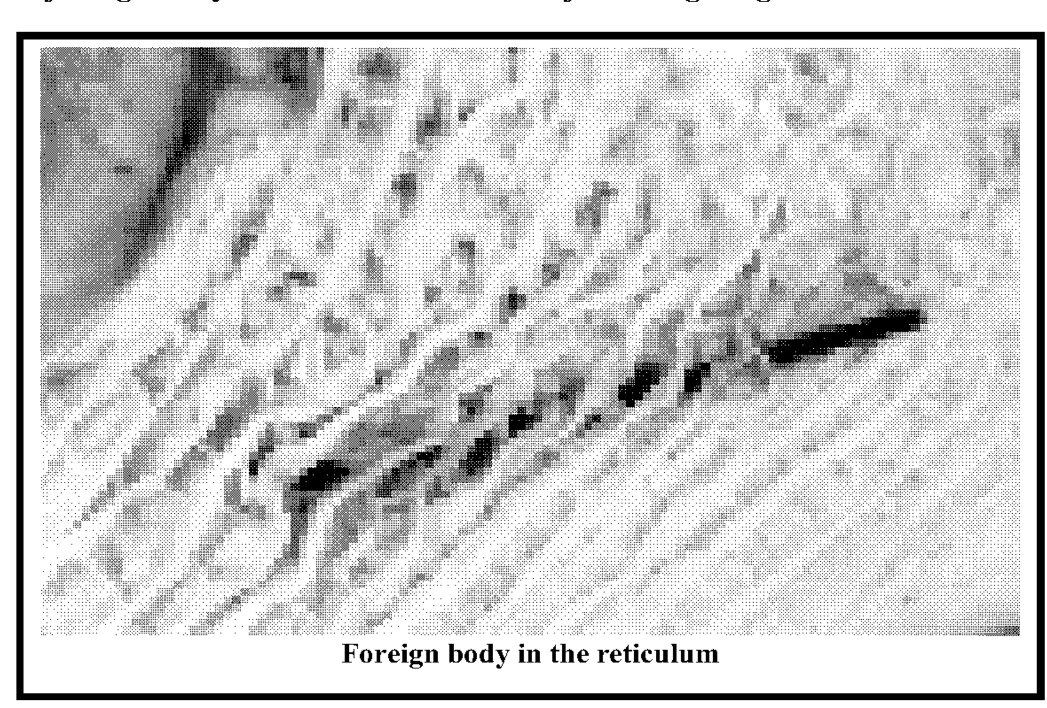
(8) Foreign body may penetrate the wall of the abomasum causing:

A- Abomasitis. B-Abomasal ulcers.

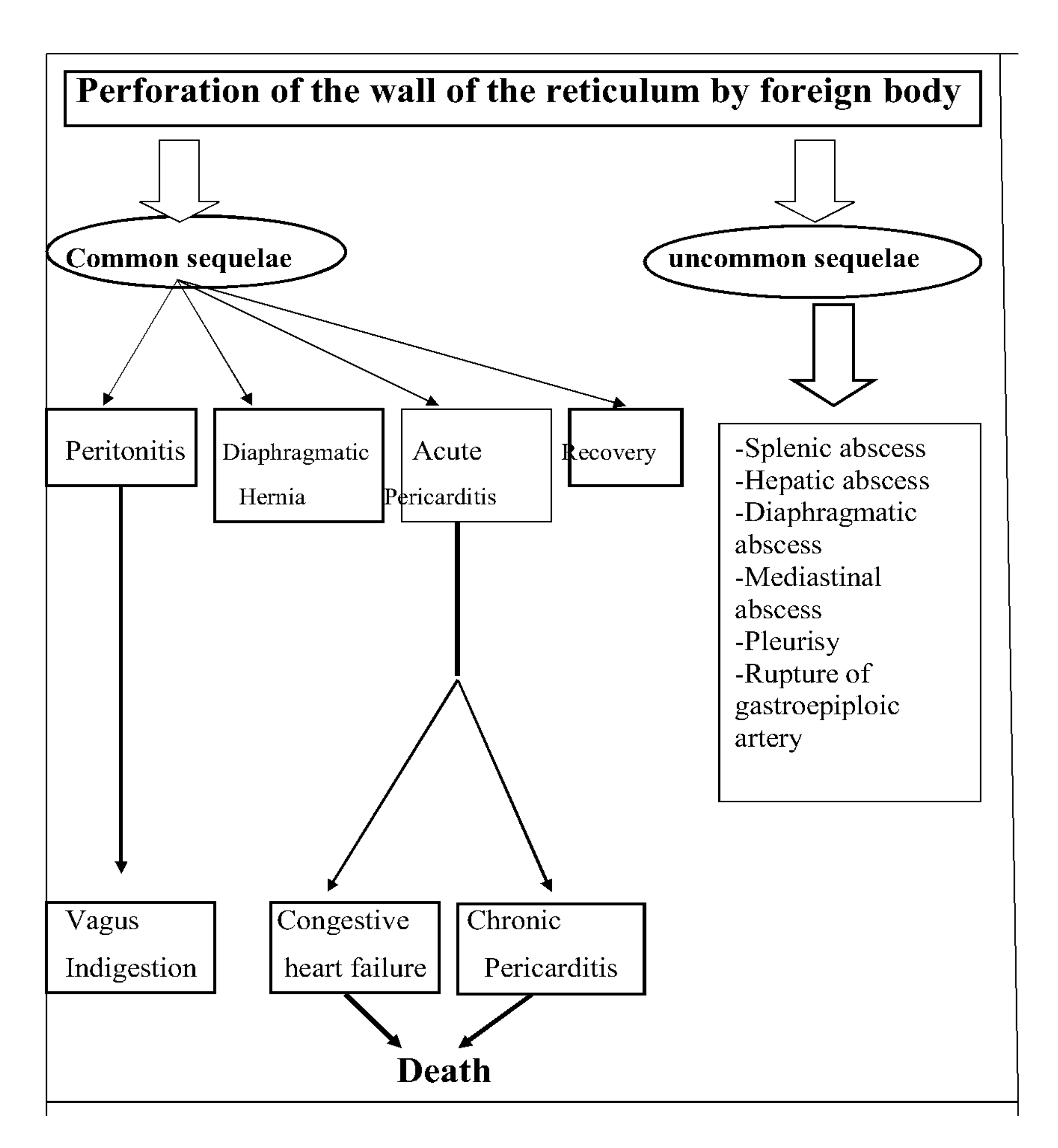
C- Pyloric stenosis that manifested clinically by anorexia and gaseous distention.

(9)Rupture of the coronary artery or ventricular wall of the heart causing cardiac tamponade.

NB. ***The complications of the penetration of the wall of the reticulum by the foreign body are summarized in the following diagram:-***



Foreign body in the reticulum


Perforation of the wall of the reticulum by foreign body
Common sequelae
uncommon sequelae
Peritonitis
Diaphragmatic Hernia
Acute Pericarditis
Recovery
-Splenic abscess
-Hepatic abscess
-Diaphragmatic abscess
-Mediastinal abscess
-Pleurisy
-Rupture of gastroepiploic artery
Vagus Indigestion
Congestive heart failure
Chronic Pericarditis
Death

Clinical Symptoms:- **in the form of :**

A-Acute local peritonitis. B-Chronic local peritonitis.

C-Acute diffuse peritonitis.

A- Acute local peritonitis:

1- Sudden onset of complete anorexia and fall in milk production to about 1/3 or less than of the previous yield and this occur within 12 hours and it's abruptly appearance is characteristic of the disease.

2- Sub-acute abdominal pain.

3- The animal reluctant to move or move slowly.

4- Arched back and the animal prefer to remain standing for long period and lie down with great care, but habitual recumbence is characteristic in other animals.

5- The animal appeared ***"gunted"*** or "***tucked up"*** due to the rigidity of the abdominal muscles.

6- Infrequent defecation and urination with the signs of pain usually with grunting the following diagram show the main common causes of grunting in large ruminant. [Constipation or scant feces is common].

(Grunting means forced expiration with closed epiglottis)

N. B. → in some cases, an attack of acute abdominal pain with kicking at belly, stretching and rolling is the earliest sign.

7- Some cases may recumbent and unable to rise.

8- Some systemic reactions may occur and varies degrees according to the degree of complications as: A-Temperature 39.5–40C° [N = 37.7–39.2C°].

B-Pulse increased up to 80/min and if there is pericarditis it may increase up to 90/min. or more.

C-Respiration 30 /min. and shallow but if there is pleurisy as a complication the respiration is painful and with characteristic an audible expiratory grunt.

9- Ruminal stases [recurrent tempany] and gaseous distention of the left flank

10- This form is short and mentioned signs can be detectable in the 1st day and it's difficult to be detected at the 3rd or 4th day.

(B) Chronic Local Peritonitis :

1- If the chronic peritonitis persist the appetite and milk yield do not return completely to the normal level.

2- Moderate chronic bloat although the ruminal movement may be normal.

3- Signs of abdominal pain are difficult to be detected but grunting may occurs during urination, defecation and rumination.

4- Constipation or dry firm feces.

5- The peritoneal fluid is increased until it may leads to distention of the abdomen in some cases and others the abdomen may appears gauntted which indication to fibrous adhesion of the peritoneum.

6-Treatment of these animals is likely to be effective or the prognosis is favorable.

(C) Acute Diffuse Peritonitis :

1-The diffuse peritonitis may develop within 1-2 days of the onset of local peritonitis.

2-Complete ceasing of the movement of the alimentary tract.

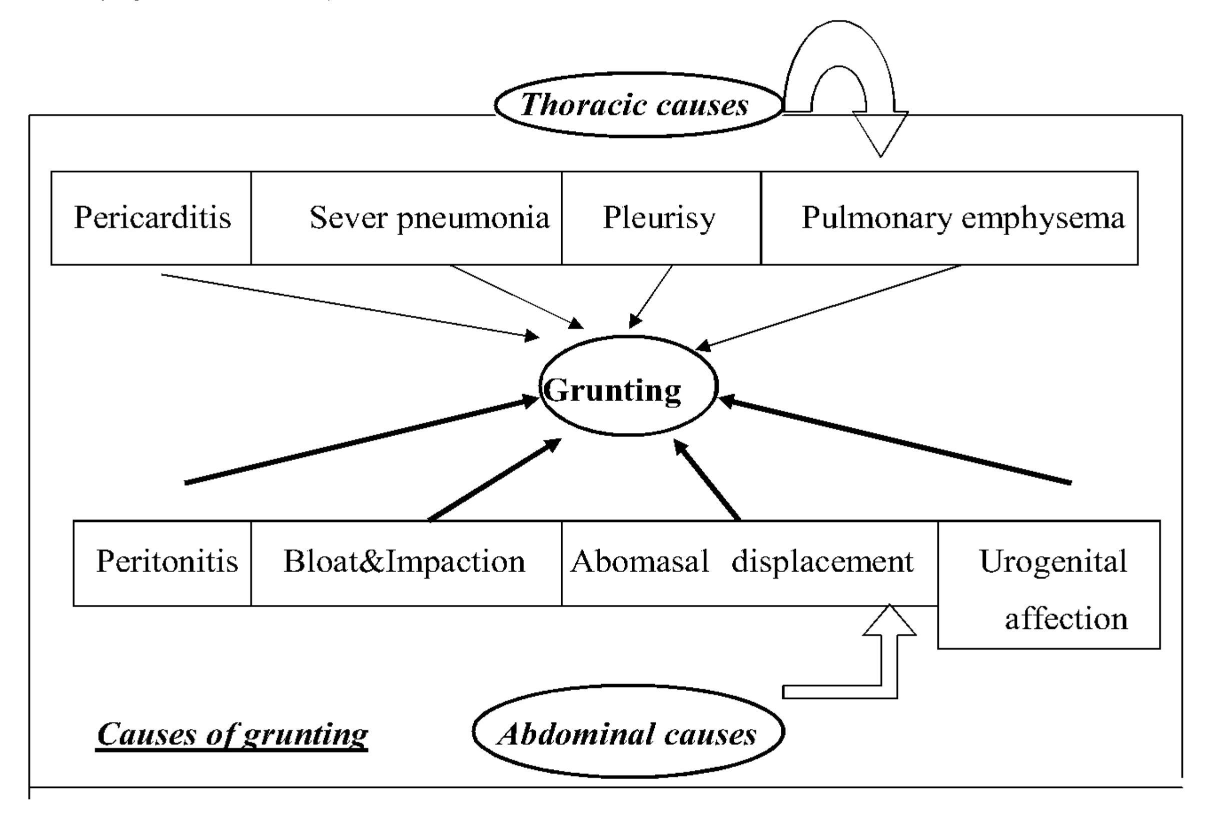
3-High body temperature or it may be sub-normal specially those cases which occur immediately after calving.

4-Pulse rate 100- 120 /min.

5-The pain can be aggravated by palpation on anywhere over the ventral abdominal wall.

6-Acute collapse and peripheral circulatory failure with absence of the reflex actions.

7-Coma and recumbence and death.



Diagnosis:-

A- History and clinical symptoms.

B- Laboratory diagnosis:- [clinical pathology]:

1-Differential leukocyte count [DLC.] is diagnostic "in chronic cases" Neutrophilia, monocytosis and moderate leukocytosis.

- Total leukocytic counts (TLC.).
- Neutrophilia up to 50-70% of T L C. [N= 30 – 65% of T. L. C.]
- Leukocytosis up to 12000 /cum. [N= 5-7000/ cum].

2- Low plasma protein concentration.

3- In acute local form → there is characteristic Neutrophilia with regenerative left shift [i.e. the number of mature Neutrophils is greater than number of immature ones].

4- In acute diffuse → there is leucopenia [T LC. below 4000 /cum] with degenerative left shift [i.e. the numbers of immature neutrophils is greater than the numbers of mature cells] which suggest unfavorable prognosis if sever cases.

5- Lymphopenia [2500-3000/cum] which indication to the stress of the infection.

6- In sever diffuse form fibrinogen may increased up to 10-20g/L.

7- Abdominal paracentesis [to detect WBCs. Protein in the peritoneal fluid– and culture against pathogens].

8- Some researches indicate that: neutronphils count greater than 40% of TLC. and eosinophils counts less than 10% of the TLC. is diagnostic of

peritonitis.

9- Radiological examination or right flank laparoscopy by using of the "flexible fibro optic laparoscope's".

10- Pain tests : Can be achieved by

A-Walking of the animal on down hill, we found the animal ascends easily but it descends with a pain and grunting.

B-Deep palpation on the ventral abdomen at the ziphoid cartilage.

C- Pinching of the weather.

D-Side stick method: By raising the animal and left it suddenly we found the signs of pain and grunting.

E-Striking by the fit of hand at the area of the reticulum on the left side [6-8th inter costal space]. We found the grunting which can be detected easily by auscultation of the trachea.

11- Metal detector now is widely used but it may give false positive with any metallic body which may by blunt or may give false negative with the non magnetic sharp foreign body as glass or plastic.

12- P. M lesions (presence of common and/or uncommon sequelae)

1- Neither acute local nor chronic local peritonitis is fatal.

2- Acute diffuse peritonitis is fatal and we found:

A-Fibrinous or supurative inflammation affect whole of the peritonium.

B- Large quantity of peritoneal fluid with characteristic odor.

13- Differential diagnosis :

1- ***Bloat or tempany*** → No fever, no sever abdominal pain

*Abruptly falling in milk yield and appetite is characteristic to reticuloperitonitis.

***2-Pyelonephritis*: -** * Presence of pus or blood in the urine (pyouria or haematouria).

3- ***Hepatic lesion*:-** *Presence of pain palpation just behind the costal arch.

*Not necessary accompanied by ruminal stasis.

4- *Acute ruminal impaction*: * Staggering and recumbence.

* Blindness due to acidity, hypothermia

*Marked increasing in the heart rate.

5-Ketosis : *By rother's test(positive Rothar`s test).

6-Traumatic Pericarditis:-

*Continue increasing in the temperature, pulse and respiration.

*Characteristic sounds of the heart by auscultation according to the stage of inflammation.

* Leukocytosis 15-30.000 /cum, eosinophilia and neutrrphilia.

7-Ephemoral fever:-* commonly occurs in outbreak form.

* Tendency to recumbence is common.

Treatment:- *A- Hygienic and conservative treatment .*

1-The feed specially roughage should be reduced to half (to reduce the contractility of the stomach).

2-Immobilization of the animal by tying of the animal in a box stall for several days.

3-Oral administration of magnet to help in immobilization of the foreign

body.

4-The ground under the forelimbs should be elevated 25 cm. than that under the hind legs to relief some sort of pain.

B- Medicated treatments : (in uncomplicated cases).

1-Intensive course of antibiotic [broad spectrum antibiotics]. For 3-5 days. With sulphonamide – but sulpha drugs is not preferred specially in lactating animals due to it has a long withdrawing time in the milk and it's residual effects in the milk and meat.(i.e. it has public health hazard when it is used for long period)

2-Anti – pyretic. Or analgesic (As Analgin-Novalgine)

3-Tonics to compensate the reduction in the food.

4-Penicillin-streptomycin combination in 3-4 liters saline or 3-4gm tetracycline intraperitoneal for 3-4 days.

Prevention:- 1-Chopped food should be passed on magnets to remove any metallic materials before being fed to the cattle.

2-Prophylactic oral administration of the magnets to be present in the reticulum and removed after slaughtering [during administration of such magnet we can give atropine sulphate 0.1 mg /kg Bwt s/c. to reduce the ruminal motility and facilitate the passage of the magnet to the reticulum.] magnets is : 7.5 cm× 1-2.5 cm diameter.

3-Tying the bales of hay by strings or rope instead of wire.

6-Vagus Indigestion

Definition:-

Vagus indigestion is a chronic disease of the fore stomachs and abomasums characterized clinically by:

1-Gradual distension of the left paralumber fossa.

2-Bilatteral distension of the venteral aspect of the abdomen due to accummulation of the ingesta in the forestomachs and abomasum.

3-Decreased appetite.

4-Weight loss.

5-Acid base imbalance.

6-Eventual weakness and recumbency.

7-Scanty feces.

8-Decrease milk production.

9-Dehydration.

-It's more common in cattle and has been recorded in sheep.

-In general the prognosis is poor but some animals may recover.

Etiology:-

1-It may be due to vagus nerve dysfunction due to injuries of the nerve as a complication of traumatic reticuloperitionitis ,as the venteral branch of the vagus nerve remified over the anterior wall of the reticulum.

2-Perireticular abscess near to the reticuomasal orifice of cattle can cause the disease [i.e.Achalasia or stenosis of the reticulo–omasal orifice].

3-Liver abscess or actinobacillosis of the rumen and reticulum may also associated with the vagus indigestion.

4-Complication of surgical interference as in surgical correction of right–sided abomasal displacement or volvalus.

5-Fibro papillomase of the cardia can mechanically occlude the distal esophagus and cause interfering with the free stomach motility.

6-Pressure on the vagus nerve in the thorax as due to tuberculous lymphnode or diaphragmatic hernia or lymphomatosis.

N.B. → Pyloric achalasia can also occur as part of a secondary indigestion due to septicemia and toxemia.

→The stomach in the ruminant has no especific nerve supply and it takes the innervation from the vagus nerve ,so any affection in the vagus nerve leads to indigestion what so called vagus indigestion

Pathogenesis : (A)

Injuries or trauma in the vagus nerve

Increase the vagal tone With bradycardia

Achalesia (stenosis) Of the reticuloomasal Orifice

Accumulation of the ingesta in the rumenoreticulum

Failure of omasal transport (Anterior functional stenosis)

Distension of the rumen with ingesta

Ruminal hypermotiality With abdominal distension (First form)

Slight degree of tempany

A neglected case leads to ruminal stasis with tempany and abdominal distension (Second form).With abomasal distension

Achalesia in the pylorus

Accumulation of the ingesta in the abomasum

Failure of pyloric outflow (Posterior functional stenosis

-Pyloric ulcer
-Abomasal distension
-Abomasal impaction

(B)-The abosamsum contains hydrochloric acid "HCL." which if not pass to the intestine due to pyloric achalasia (stenosis)it's reflux to the rumen and this known as"***Abomasal Reflux***"i.e sequesteration of the abomasal fluid to the rumen leading to increase the ruminal contentes of the chloride and potassium. leads to:-

1-Hhypocholeraemia 2-Hypokalemia

3-Hypochloremic and hypolkalemic alkalosis

4-Dehydration 5-Acid base imbalance with metabolic alkalosis

(C)-Pathogenesis of the vagus indigestion varies according to :

1-Number of branchs affected.

2-Severity of symptoms or functions disturbances.

3-Severity if trauma.

Clinical Symptoms :

1-There are 3 forms and general symptoms which characteristic of all picture

A) General symptoms B)-Ruminal distenstion with hypermotality.

C) Ruminal distention with atony.

D) Pyloric stenosis and abomasal impaction.

A)- General sympotms:-

1-Inappetance for several days.

2-An enlarging "apple" shaped abdomen [pear – shaped on the right side and apple shaped on the left side. with or without bloat.

3-The upper left half of the abdomen is distended while the

lower half of the abdomen is distended bilaterally.

4-Enlarged and impacted abomasum which can be palpated on the right flank or rectally excepte in case of advanced pregnancy.

5-Enlarged rumen which palpable by rectal examination.

6-Vital signs and responses to stimuili within the normal range.

7-No – response to treatment.

8-Scant feces [dark green].

B)-Ruminal distension with hypermotility:-

1-This type is not related to pregnancy or parturation.

2-Moderate to sever tempany although the animal is thin and not eat normally for a few weeks.

3-Fluid splashing sound may be aubidle in the ballotment of the left and right flanke if the rumen is distended with fluid.

4-Feces is normal or pasty but scant.

5-Temperature is normal with bradycardia 44-60 beats/min [N = 55-80/min.].

6-Systolic murmur is audible due to tempany or overdistension of the rumen. Causing compression on the heart and distortion of the valves (this murmur is disappeared after tempany relieved).

7-Recatally ***:A) The dorsal ruminal sac is distended to the right of the midline and is pushed back against the brim of the pelvice.***

b) The venteral ruminal sac is also enlarged and occupies much of the right lower area of the abdomen.

8-The abdomen distended in "***L-shape***" that the left flank region is distended from top to bottom and the right flank is distended only in the lower half so the abdomen take ***(L)*** shaped abdomen".

9-The adbomen is prominantly distended and the rumen is moving forcefully and almost continuosly with the sounds much reduced in volume.

10-History of failure of treatment of tempany or impaction. I.e. presence of recurrent tempany.

C)- Ruminal distension with atony:-

1-It's occurs most commonly in the late stage of pregnancy and may persiste after calving.

2-Anorexia.

3-Passing of small amount of soft pasty scant feces.

4-Distension of the abdomen.

5-History of no response to purgative, lubricants or paraympathatic stimulants therapy.

6-Ruminal movement is seriously reduced or absent with persistent mild bloat.

7-Fluid-splashing sound may be audible in the left and right. Ballotment if the rumen is distended by large quantities of fluid.

8-Normal temperature and pulse rate.

9-Rectally we found the rumen is grossly distended which may be block the pelvice inlet.

10-No-pain on deep palpation of the abdomen.

11-The animal loses weight rapidly, become very weak and eventually recumbent [in this stage the heart rate is markedly increased and the animal dies slowly].

D- Pyloric obstruction with abomasal impaction:-

1-Usually occurs in the late stage of pregnancy.

2-Anorexia with reduced volume pasty feces.

3-No. abdominal distension (No systemic reaction).

4-The distended or impacted abomasum can be palpated on the right lower quadrant as a heavy doughy viscus.

5-Rumen movements are usually completely absence.

6-Rectally the impacted abomasum can be palpated in the lower right quadrant of the abdomen as doughy viscus with pits on pressure.

7-Dehydration, electrolytes and acid – base imbalance.

8-The animal is weake and die slowly due to unthriftness .

N.B.: The ruminal distension with atony and abomasal obstruction commonly occurs in combination.

9-The distended abomasum may not be palpated rectaly or through the abdominal wall specially during late stage of pregnancy due to the gravid uterus which present in the pelvic cavity.

Diagnosis:- 1-Case history and clinical symptoms.

2-Laboratory diagnosis :

A)-Hypocholoremic and hypokalemic alkalosis.

B)-Haemoconcentration. C)-Monocytosis and neutrophilia.

D)-Ruminal fluid contains large quantities of choloride because of abomasl reflux.

PM lesiones:-

1-Empty intestine. 2-Obstructed pylorus. 3-Pyloric ulceration.

4-Lesions of traumatic reticuloperitenitis if it's increminated in the etiology.

Treatment:-

1-Empting of the rumen by using of stomach tube and flushing the rumen by using of warm water into the rumen and lavaging it by gravity flow [the contents are usually macerated and foul smelling].

2-Fluid therapy daily with oral administration of mineral oil (as parrafin oil) daily 5-10 litres.

3-Some cases may recovered after parturation but the condition may reoccure in the next pregnancy.

4-Hypertonic alkalosis.

5-Surgically interference by rumenotomy,or abomasotomy [unsuccessful].

6-Sloughtering of the diseased animal .

7-Trocare and canula may be used to reliefe the bloat.

8- Administeration of vitamine-B complex as it acts as nerve tonices

7-Left Sided Displacement of The Abomasum (LDA)

Definition:-

In this disease the abomasum is displaced from it's normal position in the right side on the abdominal floor to the left side of the abdomen between the rumen and abdominal wall.

Etiology:-

1-It's multifactorial disease.
2-Gaseous distension and hypomotality of the abomasum.
3-Feeding of high level of concentrates to dairy cattle resutt in decrease the abomasal motality and increase the accummulation of the abomasal gases.

Epidemiology:-

A) Occurrence. B) Prevalence and incidence.
C) Risk factors. D) Economic importance.

A)Occurrence:-

1-L.D.A. more common in large sized, high milk producing adult cows immediatly after parturation.
2-Approximitally 90% of the cases occur 6 weeks after parturation but some cases may occur several weeks before parturation. i.e. late stage of pregnancy.
3-More common in loose housing and feeding on grain system. i.e. no grazing.

4-More common in winter season which may be areflection of either a higher frequency of calving or relative inactivity of the animal .

5-The disease is recorded in 8-14 weeks of age in calves and 21 months old primigravide heifer.

B- Prevelence and incidence:-

LDA. is more incidence than right sided displacement of the abomasum.

C-Risk factors:-

1-Feeding on large amounts of grain as corn and corn silage acts as predisposing factor,so the disease is more common in the high producing animals as they are fed on large quantities of grains.

2-Crude fiber concentration in the diet of dairy cows less than 16-17% considered as a riske factor for left sided displacement of the abomasum.

3-Heavy grain feeding is though to increase the flow of the ruminal ingesta to the abomasum which causes increase the concenteration of the volatile fatty acids which may inhibite the motality of the abomasum and leads to LDA.

4-Low motality of the abomasume lead to accummulation of large amounts of ingesta which not pass to the doudenum ,also causes accummulation of gases as methan and carbone dioxide that leading to abomasal distension and it's displacement.

5-Late stage of pregnancy considered as a significant risk factor due to in this period the rumen is pushed from the abdominal floor by the

expanding uterus and the abomasum is pushed forward and to the left under the rumen causing LDA.

6-Hypocalcemia also considered as predisposing factor due to the muscular atony of the stomach that associated with hypocalcomic cases.

7-Females are more susceptable than males specially heavy dairy cattle than beef cattles.

8-Genetic factors may be suggested.

9-Experimentally metabolic acidosis may leads to abomasal atony and decrease the emptying rate of the abomasum which act as a predisposing factors of LDA. Also, vitamin A deficiency may acts as a predisposing factor of LDA.

D-Economic importantce :-

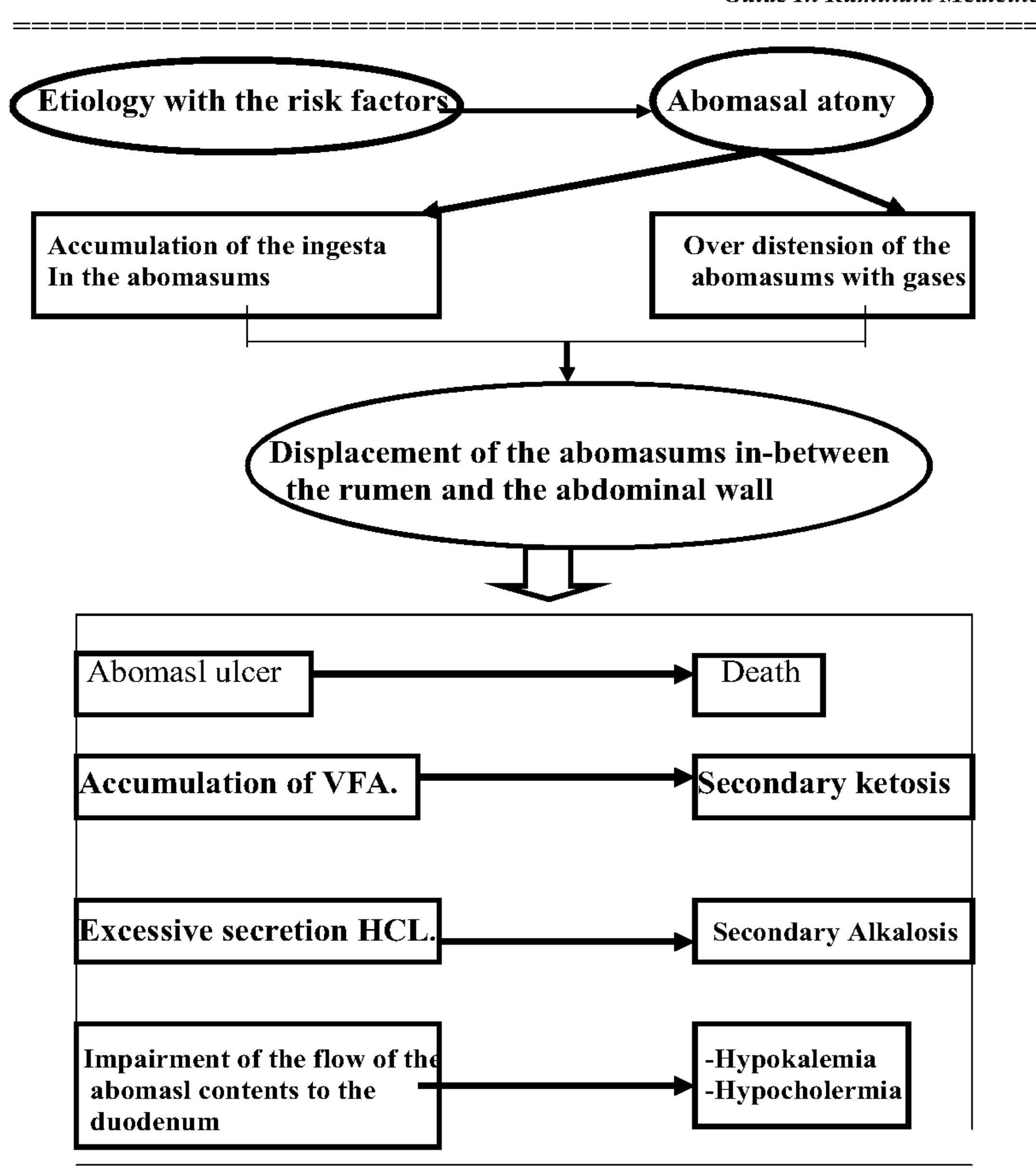
1-High coast of surgery

2-Usually associated by low in milk yield

N.B.:In occosional cases the abomasum become trapped anteriorly between the reticulum and diaphragm called "***Anterior displacement of the abomasum***".

Pathogenesis:-

Digaram of the left sided abomasl displacement in the following page



Pathogenesis of Left Sided Abomasl Displacement

Clinical Findings:-

1-Inappetance to sometimes almost complete anorexia usually within few days or week following parturation.

2-Marked drop in milk production. – varying degree of ketosis

3-On visual inspection the left lateral abdomen is "***slab-sided***" because the rumen is smaller than normal and displaced medially.

4-Temperature, respiration and pulse rate are usually within normal ranges.

5-The Feces is reduced in volum but softer than normal but periode of profuse diarrhoea may occur.

6-Ruminal movement are present but decreased in frequency and intensity until the sounds are not heard.

7-Auscultation on 10-12th left intercostal space we found high pitched tinkling or splashing sound which is the sound of abomasum (one sound per 15 minutes)and such sound is stimulated by tactile percussion [Normally the abomasum sound can be heard at 7-9th right intercostal spaces].

8-Some cases reveal sudden onset of anorexia with signs of abdominal pain and distension. [these are acute cases and not more common].

9-An obvious bulge caused distended abomasum may develop in the anterior part of the upper left paralumbar fossa and this may extend up behind the costal arch almost to the top of the paralumbar fossa.

10-In acute cases the temperature may reach 39.5C and the pulse rate up

to 100/min.

11-The appetite may returne to the normal level but it's selective i.e. the animal eating only certain food particulary hay [fibres].

12-In rectal examination we can detect tympanic rumen but abomasum can not be detected rectally in case of LDA.

13-In untreated case the animal become sever inanition and remain static for long period and may be die.

14-The fat animal may show signs of ketosis or fatty liver syndrome.

15- *In anterior displacement of the abomasum* :

*The signs is very similar to that mentioned above except that normal ruminal sounds can be heard in the usual position and gurgling sounds charactristic of a distended abomasum are heard just behind and above the heart and on both sides of the chest.

16-Atrial fibrillation may be present in some cases which is considered to be caused by a concurrent metabolic alkalosis which uasally associated with the pathogenesisi of tLDA (See the pathogeneisi),these sign is disappeared immediately after surgical correction .

17-*In calve* there are:- -Inappetance - Loss of body weight.

- Recurrent distension of the left paralumbar fossa.

-Metalic ping sound and fluid splashing sounds on auscuttation and percussion of the left flank region.

Diagnosis :- A)- History and clinical symptoms.

B)-Laboratory diagnosis :

1-There is no particular change in the blood picture unless there is inter-current disease particulary traumatic reticuloperitenitis or abomasal ulcer.

2-Ketonuria [moderate to sever degree] but blood glucose level is within the normal range.

3-Haemoconcentration which evidenced by (increase PCV- increase Hb – Increase total serum protein).

4-Mild metabolic alkalosis 5- Hypochloremia. 6-Hypokalemia

7-Hypocalcemia.

C)-Differential diagnosis : 1-Traumatic reticuloperitenitis.

2-Diaphragmatic hernia. 3-Ketosis. 4-Vagus indigestion.

Treatment:- 1-Mainly by surgical correction of the position of the abomasum. 2-Vigrous rolling of the animal.

3-Violent exercise may lead to spontonouse recovery

4-Parenteral glucose and oral propylene glycol is used to avoid ketosis and fatty liver syndrome.

5-Liquid parafin upto 2 liters . 6-Reliefe the tempany.

Control:- 1-Feeding of large quantities of roughage in approach to parturation.

2-Avoiding overfeeding on grain specially in late stage of pregnancy.

3-Avoiding any alteration in feeding system near to the parturation which may lead to indigestion which acts as a presdisposing factor of LDA.

4-Slight excercising in the prepartum period specially in close housed cow.

8-Right Sided Displacement Of Abomasum (RDA) And Abomasal Volvulus

Definition:-

RDA is a case in which the abomasum is displaced to the right side of the abdomen between the liver and abdominal wall it occurs commonly in the mature dairy cow a few weeks after parturation and it's charactrized clinically by :

1-Absence of rumination.

2-Depression.

3-High pitched metalic sound during auscultation over the right flank region.

4-Scanty abnormal feces.

5-Gradual distension of the right side of the abdomen due to accumulation of the fluid and gas in the abomasum.

-RDA is aprecurser -but not necessary - to abomasal volvulus which is an acute obstruction of the alimintary tract which charactrized clinically by:-

1-Complete anorexia.

2-Abdominal pain.

3-High pitched metalic resonant ping sound on percussion and auscultation of the right flank with fluid splashing sound.

-Prolonged abomasal volvulus leads to accumulation of fluid in the abomasum causing hypokalaemia, hypochloremia, metabolic alkalosis

with dehydration and shock.

Etiology:-

-Similar to that of left sided abomasal displacement.

Incidence and occurrence :

1-More common in mature cow 3-6 weeks after calving.

2-More common in high producing cow specially that fed on large quantities of grain and indoor winter fed animals.

3-It may occur in calves after some weeks of age upto 6 months of age without previous history of illeness which suggest that the cause is accidentaly.

4-The incidence increased with advanced age and the risk is high with advanced age 4-7 years of age of dairy cows.

5-It's less common in bull and pregnant cow.

Risk factors :

1-Similar to that of L.D.A.

2-Correction of LDA. may lead to abomasal volvulus due to casting or rolling of the animal.

Pathogenesis :

A)-Etiology and predisposing factors lead to :-

1-Abomasal atony

2-Accummulation of the ingesta in the abomasum.

3-Gradual distension and displacement of the abomasum in acuadal

direction on the right side for several days [dilatation phase] and in this phase there are :-.

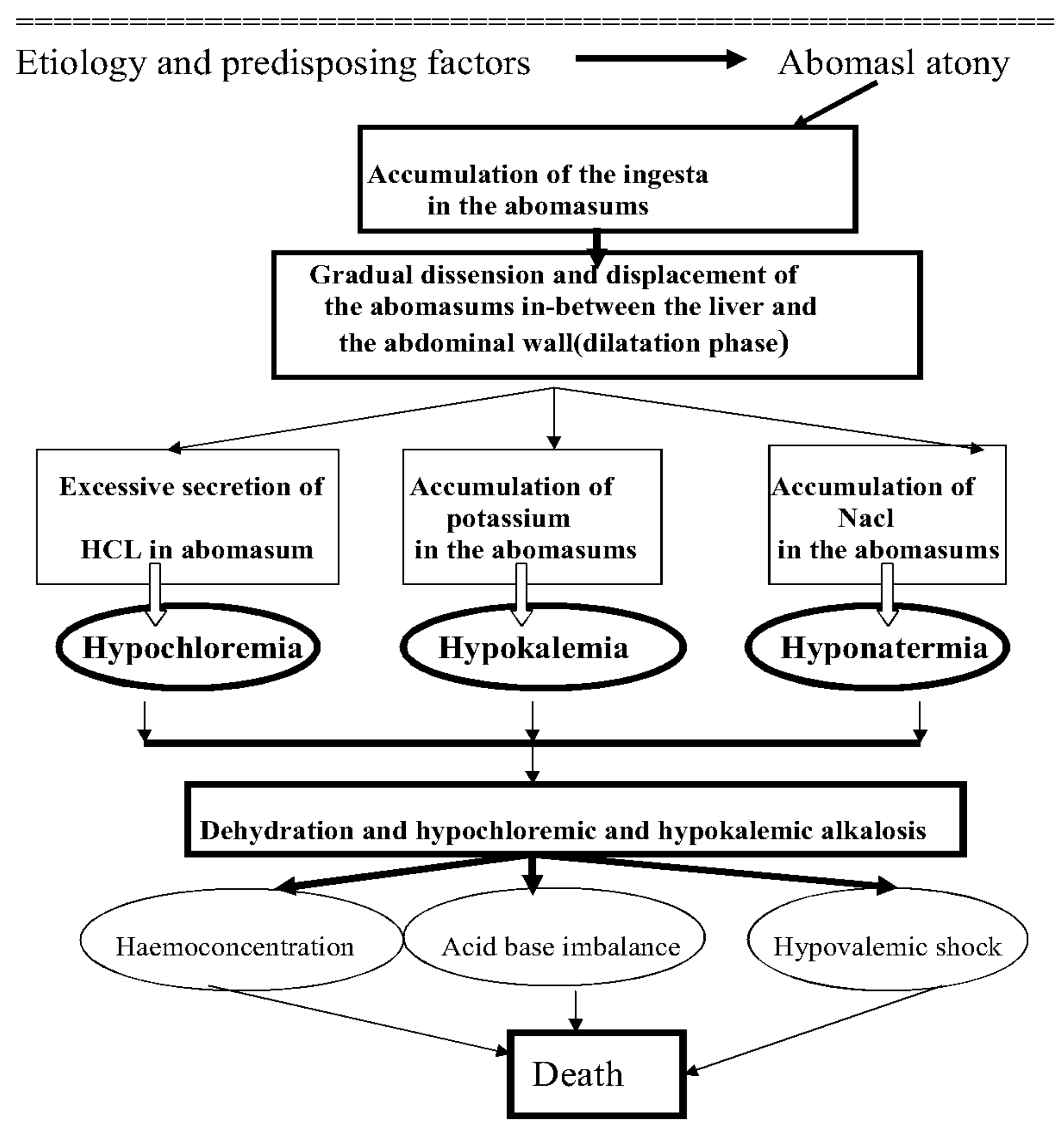
I)-Dehydration and hypochloremic and hypokalemic alkalosis

II)-Upto 35 litres of fluid may accummulated in the dilated abomasum of mature 450 kg cow resulting in dehydration which will vary from 5-12% of body weight.

III)-The degree of dehydration can be taken as prognostic aid prior to surgical interference.

B)-RDA. may lead to compression on the cuadal vena cava causing stimulation of the sympathatic nerveous system causing tachycardia

C)-Ddilatation phase may followed by twisting or volvulus of the abomasum in clockwise or anticlockwise direction causing ischemic necrosis and impairment of the circulation of the abomasum and may compress or may lead to rupture of abomasum followed by peritonitis and sudden death.



Pathogenesis of Right Sided Abomasal Displacement

Clinical Symptoms:-

1- In right sided dilatation or displacement of the abomasum there is usually a history of A)-Calving within the last few weeks

B)-Inappetance and decrease in milk production for long period.

C)-Abnormal feces and reduced in amount

2-Anorexia usually complete when the abomasum is dilated & distended.

3-Depression, dehydration, increased thirst and no interest in feed.

4-Respiration and temperature usually within normal range.

5-Musculare weakness and the heart rate may be normal or upto 100/min.

6-The mucous membranes are usually pale and dry.

7-Ruminal stasis and the ruminal palpation is excessively doughy.

8-The distended abomasum easily to be palpated immediately behind and below the right costal arach.

9-Ballottement of the middle third of the right flank immediately behind the right costal arch along with simulterous auscultation will reveal fluid splashing sounds suggesting a fluid filled viscus.

10-In advanced cases the abdomen appeared wholy distended specially the right flank region.

11-In sever case the cow may recumbent with agrossly distended abdomen and grunt with each respiration(see the causes of grunting in reticuloperitonitis).

12-Rectal palpation is very important :

A)-In case of dilated phase the abomasam is partially palpated.

B)- In case of abomasal volvulus it can be palpated as distended tense viscus and it is usually palpated in the right flank anywhere from the upper to the lower quadrant.

13-The feces are usually scant, soft (not confused with diarrhoea) and dark in colour.

14-Animal suffering from abomasal volvulus usually recumbent 24 hr. after volvulus and death occurred with in 48-96 hours after volvulus due to sever dehydration, shock or rupture of the abomasum which causes sudden death.

15-In calves with acute abomasal volvulus there are :

A)Abdominal pain with kicking of the belly.

B)-Sudden onset of anorexia.

C)-Depression of the back.

D)-Belowing and straining.

E)-Auscultation of right frank region usually reveal high pitched tympanic sounds.

F)-Pain on palpation immediately behind the right costal arch.

G)-Heart rate usually 120-160/min.

16-In some cases there is profuse diarrhoea and the feces may become blood stained .

Diagnosis:-

A)-History and clinical symptoms :

B)-Laboratory diagnosis :

1- Increase of PCV and total serum protein
2- Presence of Alkalosis. Hypochloremia and Hypokalaemia
3- In late stage of volvulus there are leukopenia, neutropenia due to ischemic necrosis of the abomasum and peritonitis.
4- Paracentesis of the distended abdomen reveals that fluid without protozoa with pH 2-4 and the fluid may be serosanguinous when torsion of the abomasum is present.

C)-:Differential diagnosis:-

- The high pitched metallic ping sound in case of abomasal displacement (right) is heared only in the 9^{th}-12^{th} intercostal spaces not extended to the right paralumber fossa. While in case of abomasal volvulus it's more extended cranially and caudally and in the right panalumber fossa but not completely filling the fossa.

Abomasal impaction : -

-The rumen also impacted and distended.

-The abomasum is usually palpated firm or doughy and it's present more in the lower part of the abdomen while in case of RDA it's usually present in the dorsal part of the abdomen near to the right paralumber fossa.

-There is no tympanic sound as that of RDA.

Chronic or sub – acute reticuloperitonitis there are :

- Mild fever. – Positive pain test
- Grunting on deep palpation of the abdomen .
- Guntted abdomen. - Dry firm feces.

Prognosis:-

Prognosis of right sided displacement of the abomasum is favourable if the diagnosis is made within a few days after the onset of the clinical sign or before accummulation of large quantities of the fluid in the abomasum.

Treatment:-

A)-Medicated treatment in milde case :

1-500ml of 25% calcium borogluconate interavenously to improve the abomasal motality.

2-Feeding on good quality hay but no grain for 3-5 days and the animal is monitored daily.

3-Electrolytes therapy for correcting the metabolic alkalosis and dehydration and some cases may not requiered to surgical correction and it may returned to normal within 2-3 days.

4-Mineral oil as : caster oil or parrafin oil (5-10 liters /day) or magnesium hydroxid [500gm/2days] to evacuated the abomasal contents.

5-Fasting may give good result (recommended based on field experience).

6-The severly advanced cases need surgical correction of the abomasum by laparotomy.

7-Fluid can be used as source of potassium, chloride and sodium (KCL 108gm+ NaCL 80gm + H_2O liters) to correct alkalosis.

B)- Surgical correction of the abomasum

Peritonitis

Definition:- Peritonitis means that inflammation of the peritonium which characterized clinicaly by abdominal pain which varies according to the severity and extent of inflammation, fecal stasis, toxemia and tenderness on palpation and rigidity of the abdominal wall.

Etiology:- It may be primary or secondary/

1-Rupture of the abomasum after torsion or volvulus.

2-Ruminitis as sub-sequence of carbohydrate engorgement(lactic acidosis).

3-Hepatic or splenic abscess.

4-Rupture of the vagina in the young heifer during violent coitus with a young active bull.

5-Chemical peritonitis due to injectionn of hypertonic solution intrapertonialy as calcium preparation in treatment of milk fever.

6-Surgical fautls as in case of correction of LDA. or rumenotomy.

7-Interaperitoneal injection of non-steril solution.

8-Spontenous rupture of the uterus during dystocia or parturation.

9-Spontenous rupture of the rectum at calving.

10-A part of some specifice diseases as tuberculosis

11-Etiology or as sequalea of traumatic reticuloperitonitis.

12-Traumatic perferation of the abdomen from extrior.

13-Faulty asepsis during :-

A)-Laparotomy B)-Intraperitoneal injection.

C)-Trocarization for tympanic rumen or caecum.

14-Ruptured abscess in any abdominal viscus.

Paathogenesis:-

At least there are six (6) factors which are responsible for genesis of the charactristic clinical features of the peritonitis. These factors are :

A-Toxemia. B-Shock and haemorrhage.

C-Paralytic ileus. D-Adhesion.

E-Accummulation of fluid and exudate. F-Abdominal pain.

A)-Toxemia:-

1-The toxins which produced either by (bacteria or destroide tissue) are absorbed rapidly by the peritenium causing toxemia.

2-Ruptured of part of alimentary tract or in the urogenital tract and spillage of large quantities of ingesta in the peritoneal cavity causing sever toxemia and acute peritonitis which may cause sudden death within 2-3 hourse after rupture .

B)- Shock and haemorrhage:-

Shock usually due to sudden deposition of the gut contents or uterin contents in the peritoneal cavity pluse the haemorrhage which resulted from rupturing of the gut, uterus or rectum or other organs.

C)- Paralytic ileus : Occurs due to:-

1-Reflex inhibition of the alimintary tract tone and movement in case of acute peritonitis.

2-Intestinal obstruction or surgical interference which also may leads to rupture of the gut.

D)- Adhesion:-

1-Trauma to the peritonium leads to production of serosanguineous. exudate which contains two closely bound proteins which are :

A)-Fibrinogen. B) Plasiminogen.

2-Fibrinogen by the thrombin is converted into fibrin which leading to formation of fibrinous adhesion of the inflammed part of the peritonium this adhesion has an important role in.-

A)-Localizing the infection and inflammation.

B)-Play an important role in the healing process.

But : this adhesion has bad effect as it :

A)-Causing functional or mechanical obstruction of the gut.

B)-Interfere with the normal motility of the gut.

C)-Vagus indigestion.

3-The animals vary in their own amount of the fibrinogen as the following:

****In cattle:***

The percentage of fibrinogen is high which response rapidlly to the traumatic peritonitis by fibrinous adhesion and localizing of the inflammation,so cattles are less suciptable to diffuse peritonitis.

***In horse** :*

The percentage of fibrinogen is low, so horses are highly susciptable to diffuse peritonitis(fatal) that due to lack of the role of the adhesion (which done by fibrin) to localizing of the inflammation.

4-Plasminogen is converted into plasmin by plasminogen activatore which secreted from the mesothelial cells.

5-Plasmin has fibrinolytic effect that favoring lysis of the early adhesion.

E)- Accummulation of fluid and exudate :

From the ruptured organ(s) which may lead to :-

A)-Abdominal distension.

B)-Interference with respiration by obstruction of the normal diaphragmatic movement.

F)-Abdominal pain:

Due to inflammation of the serous surface of the peritonium which sufficient to produce abdominal pain.

Clinical Finding:

A)- Acute and subacute peritonitis :

1-Inappetance and anorexia.

2-Toxemia and fever :

3-The temperature reach 39.5C for 24-36 hours in acute local peritonitis while in acute diffuse peritonitis there is high or sever fever upto 41.5C but in terminal stage the temperature may falls to sub-normal temperature.

4-Increase the pulse rate and respiration with grunting sound with the end of expiratory movement.

5-The feces are scanty, soft or may be diarrhoea and it may contains mucus, the feces may be absent for period upto 3 days.

6-Ruminal contraction is reduced or absent in acute form while in chronic form the ruminal movement may be present but reduced than normal.

7-Abdominal pain which evidenced by:

a)-Disinclination to move or to lie down.

b)-Lying down with great care and grunting with pain.

c)-Arched back and the gait is stambling and cautious.

d)-Grunting with defecation and urination .

8-Sever abdominal pain in deep palpation of the abdomen.

N.B.: In case of peracute diffuse peritonitis which usually occurs after calving due to repture of the uterus or rectum the animal characterized by :

1-Depression. 2-High pulse rate upto 120/min.

3-Sub-normal temperature. 4-Death within few hours after parturation.

5-Fibrinous adhesion can be detected by rectal examination.

B- Chronic peritonitis :

1-The development of adhesion which interfere with the alimentary tract movements, and gradual spread of the infection as the adhesion break down, combine to produce a chronic syndrome of indigestion and toxemia which punctuated by short, recurrent attack of more sever illness.

2-Partial obstruction of the intestine may occurs with gaseous distension.

3-Local adhesion can be palpated by rectal palpation.

4-The course of chronic peritonitis is longe and the prognosis is unfavourable.

5-Weight loss is sever.

6-Intermittent episode of abdominal pain suggesting intestinal colic.

7-Sub –cutenous edema of the ventenal abdominal wall may be present in some cases.

Diagnosis:-

A)-Diagnosis of peritonitis is difficult due to the predominant clinical symptoms are often more common to other diseases.

B)-History and clinical symptoms.

C)-Laboratory diagnosis or clinical pathology :

1-In acute diffuse peritonitis -Leukopenia

-Neutropenia -Marked increase in immature neutrophils

2-In local form : Similar to that of traumatic reticuloperitonitis

3-The level of plasma fibrinogen is increased according to the degree of the peritonitis.

4-Examination of the peritoneal fluid taken by abdominal paracentesis is a good diagnostic aid to detect the specific anti-microbial drugs that can be used in treatment [microbiological examination by sensetivity test].

N.B.: In the peritoneal fluid sample if :-

(I)-Presence of ingesta in the peritoneal fluid sample during paracentesis is

indication to intestinal ischemic necrosis or rupture.

(II)-If there is blood this indication to damage of the wall of the viscus.

(II)-Presence of pus or leukocytes this indication to inflammation.

(IV)-Presence of urine this indication to rupture of urinary bladder or urethera.

PM lesions:-

1-In case of acute diffuse peritonitis the entire peritonium is involved.

2-The most sever lesions present in the venteral abdomen.

3-Presence of fibrin adhesion which easly broken down.

4-Haemorrhage into the subserosa.

5-In cattle corynebacterium pyogen and fusebacterium necropherum are present in large numbers and produce a typical, nauseating odor.

6-Presence of etiological factors as ruptured vagina, uterus or presence of ***PM. lesions*** similar to that of the traumatic reticuloperitonitis.

Treatment:-

1)-Treatment the specifice causes of the disease.

2)-Chronic form less responsive due to the serious involvement of the gut with fibrous adhesion.

3)-Non-specifice drugs as broad spectrum antibacterial drugs and sulphonamide can be given orally or parentarly or interaperitoneally injection with isotonic non-irritant solution.

5)- Sloughtering the animal specailly in case of rupture of vital organ as uterus.

6)-Treatment of toxemia.

E- Diseases Of The Liver

*Moste of the liver diseases in the ruminants are manifested by the following symptoms in general :-

1- Jaundice.
2- Nervous signs [Hepatic encephalopathy].
3- Odema and emaciation (ascitis).
4- Abdominal pain.
5- Diarrhoea and constipition in alternative form.
6- Photosensitization [phylloerythrin].
7- Haemorrhagic diathesis.
8- Alteration in size of the liver (increased in size or decreased in size).
9- Displacement of the liver.
1o-General weakness and emaciation

Hepatitis

Definition:- *Hepatitis means that inflammation of the liver which may be local or diffuse and it may leads to fibrosis or cirrhosis of the liver.

Etiology:- ***According to the types of hepatitis :***

A)-Toxic Hepatitis:-

1-Inorganic poisones as:-copper–phosphorus–arsenic and selenium toxicity.

2-Organic poisones as:- carbontetrachloride or chloroform Plant poisons

3-Misceleneous farm poisones or chemicals as : (dried poultry wast- cotton seed cake ……etc.).

4-Toxemia may lead to hepatitis in dairy cattle after metritis or mastitis due to endotoxemia.

5-Hepatitis also may occurs after extensive damage of the body tissues or after burns, injuries or infarction also due to toxemia.

N.B.: The lesions in case of toxic hepatitis usually centerilobular and it may be mild in degree which manifested by cloudy sowelling or in sever form accompained by extensive necrosis which may lead to fibrosis of the liver.

B- Infectious Hepatitis:-

1-Rift vally fever. 2-Viral rhinopneumonitis (herpes virus.)

3-Chlamydia spp. 4-Histoplasmosis.

5-Clostredium Novyai (infectious necrotic hepatitis.).

C- Parasitis Hepatitis:-

1-Chronic or acute liver fluke (fascioliasis).

2-Migrating larvae as in in case of Ascariasis.

D- Nutrotional Hepatitis:-

1-Methionin deficiency. 2-Vit. E and selenium deficiency.

3-Cystien deficiency (hepatic necrosis).

4-Cobalt deficiency (white liver disease).

E- Congestive hepatopathy:-

-Usually due to congestive heart failure which may leads to centerilobular degeneration of he hepatic tissues.

N.B.:1-Hepatitis may occurs related to heriditany factors.

2-There are some diseases give hepatitis but not give the signs of hepatitis during the life i.e. we found some liver affections or lesions as necrosis in postmortum examination but it is not show the clinical signs of hepatitisas in case of:- A)-Salmonelosis. B)-Listeriosis.

C)-Chemical hepatitis or hepatic abscesses due to perforation of the rumen due to impaction or rupture of other organs or extension from peritonitis. D)-Leptospirosis.

Pathogenesis:-

A- Toxic Hepatitis :- The effect of endotoxin on the liver include multifocal hepatocellular necrosis which leads to:-

*Decrease the hepatic glucogenesis. *Decrease the hepatic blood flow.

* Stimulate the kupffer cells to secrete lysomal enzyme – prostaglandin and collaginase that leading to excessive damage of the hepatecytes and in advanced cases may cause liver cirrhosis or fibrosis.

B- Infetious hepatitis:- The lesion may be focal or diffuse according to the causitive agent.

C-In parasitic hepatitis.-

*The lesions vary according to the types and numbers of migrating larvae.

Clinical Symptoms:- 1-Muscular weakness – jaundice.

2-Anorexia – mental depression – excitement in some cases.

3-In the terminal stage there is sommolence, recumbency and coma with

intermittent convlusions.

4-Photosensitization with a break of the hair leading to shedding of the coat.

5-The anorexia may be associated with constiption and punctuated by attack of diarrhea.

6-The feces is lighter than normal in colour

7-Nervous signs in the form of → Mania and aggressive behavior.

→ Tremors, yawning and ataxia.

N. B: Odema due to failure of the liver to anabolize amino acids and protein ceusing fall in plasma protein and low osmatic pressure of the plasma which are sufficient to causes odema.

8-The late stage charactarized by what so called ***dummy syndrom:***

A)-No response to normal stimuli.***B)***-Blind ness and pushing with the head.

1- Abdominal pain.

9-The signs of hepatic fibrosis similar to that of hepatitis but it's developed more slowly and persiste for long period with odema or ascitis and jaundice (as in case of bottle jaw in chronic fascoliasis).

10-In young animals there is stunted growth, ascitis and hepato-encephalopathy.

Diagnosis:- **A)-**Case history and clinical symptoms.

B)-Clinical Pathology :

1-By detection of liver function test : GOT(AST),GGTand GPT(ALT)

2- Liver biopsy

C)-P. M. lesions:- 1-Congested, enlarged and swollen edges of the liver. 2-The liver may be pale in color.

3-Presence of tracks and sub-capsular haemorhage in case of parasitic heaptitis.

4-Necrosis and fibrosis of the hepatic tissues may be present.

5-Fatty infitteration of the hepatic paranchyma.

6-Signs of jaundice.

Treatment:-1)-Firstly feeding on ration rich in carbohydrate and calcium and low in protein and fat that due to damage liver failure to detoxify the ammonia and other nitrogenous substances [high level of ammonia due to failure of hepatic detoxification leads to nervous signs].

2)-Administration of broad – spectrum antibiotics has been introduced to control protein digestion and putrifaction and to prevent the secondary bacterial infection ,the best are. Neo mycin and choramphencol we found disappearance of hepatic coma and depression of blood ammonia level with the using of this antibiotices.

3)-Adminestration of vitamins are desirable specially vitamine B-complex.

4)-Hepatic fibrosis is considered to be a final stage in hepatitis and treatment is not usually undertaken.

5)-Treatment of the caustive agentas in case of facioliasis by giving of he suitable anthilmentic drugs.

6)-Using of the vacinal rogram against infectiuos diseases.

Chapter No (2)

Cardiovascular System

1-Traumatic Pericarditis

2-Congestive heart failure

3-Acute heart failure

4-Peripheral cirdulatory failure

5-Pericarditis

6-Venous thrombosis

7-Myocardial diseases

8-Endocarditis

9-Valvular diseases

1-Traumatic Pericarditis

Definition:-

It's perforation of the pericardial sac by sharp foreign body originating from the reticulum causing pericarditis with development of toxemia and congestive heart failure, characterized clinically by: Tachycardia – fever – engorgment of jugular vien – anasarca - hydrothorax – ascitis and abnormalities in the heart sounds are the diagnostic features of the disease.

Etiology:-

1-Usually similar to that of traumatic reticuloperitenitis i.e. traumatic pericarditis usually a sequalea of traumatic reticuloperitenitis.
2-Usually or more common in pregnant animal.

Pathogenesis:-

1-Enterance of sharp foreign body by ingestion and lodged in the reticulum and by the reticular movement it may perforate the reticular wall and diaphragm then to the pericardium.
2-Introduction of mixed bacterial infection from the reticulum with the foreign body leads to :
3-Sever local inflammation and toxemia and local hyperemia leading to frictional sound.
4-Accumulation of fluid in the pericardial sac and pressure on the heart causing CHF(congested heart failur) with drippling sound.
5-In the late stage the peri-cardial sac may be effused with the

pericardium with organization of the exudate with the formation of fibrous tissues with muffeled sound and adhesionof the pericardial layers and death.

6-Traumatic pericarditis may be associated with pulmonary congestion or odema due to the pulmonary hypertension which resulted from the congestive heart failur.

Clinical symptoms:-

1-Depression with complete loss of apetite (anorexia), hapitual recumbency with fever 40-41C.

2-Rapid body weight loss and tachycardia upto 100/min (N =55 –80/min).

3-Diarrhoea or scant feces may observed.

4-Nasal discharge due to pulmonary odema.

5-Engorgment of the juagular vien with visible juagular vien pulsation.

When we make puncture or I.V. injection of the juagular vien we found :-

-The blood come through the needle in jets.

-There is resistence during injection due to rigidity of the vien.

-Cord like vien.

6-Arched back with abducted elbows.

7-Shallow rapid respiration 40-50/min (N =15-30 /min) and it's wholly abdominal and accompained by grunting .

8-Odema in the brisket or dewlabe and in the venteral aspect of the abdomen.

9-Ruminal movements are present but depressed.

10-Recurrent tempany and vagus indigestion may be present as a result of previous peritonitis .

11-Dyspnoea which may leads to asphyxia and death.

12-Death occurs within 1-2 weeks due to toxemia or asphyxia or C.H.F.

13-During percussion : we found increasing in the area of cardiac dullness.

14-On Auscultation : we found abnormalities in the heart sounds according to the stage of the disease as the following :

1st stage: Frictional sound due to hyperemia and congestion in the pericardial sac.

2nd stage : Dribbling or succsion sound due to presence of suffucient amount of fluid or exudate in the pericardial sac.

3rd stage : Tinkling or splashing sound due to presence of gase with the exudate in the pericardial sac.

4th stage : Muffulled sound due to effusion or adhesion of the pericardial sac and organization of the exudate with the formation of fibrous tissues in the pericardium.

15- Positive Pain test [see reticuloperitionitis].

Diagnosis.-

A)-Case history and clinical symptoms are characteristic.

B)-Laboratory diagnosis :

1-Leukocytosis 16000-30000/cum^2 [N =4 –12 thousands]

2-Neutrophillia and eosinopenia.

C) PM lesiones :-

1-Gross distension of the pericardial sac with foul smelling and grayish fluid containing flaks of fibrin.

2-Serous surface of the pericardial sac carries very heavy deposits of newly formed fibrin.

3-Presence of cord like fibrous sinus tract usually connect the reticulum and pericardial sac(along with the pathway of the penetrating foreign body).

4-Presence of PM lesiones similar to that of traumatic reticuloperitonitis

5-In chronic cases the pericardial sac is thickened and fused to the pericardium by strong fibrous adhesions with pus or thin straw coloured fluid.

Treatment:-

1-As mentioned before in the treatment of traumatic reticuloperitonitis

2-If the animal pregnant we give intensive course of anti-biotice until parturation then sloughtering of the animal.

3-Surgical interference may be used (unfavorable or with unpredectable prognosis).

2-Congestive Heart Failure (CHF)

Definition:- Congestive heart failure is a condition in which the heart is unable to maintain circulatory equilibrium at the rest condition and the congestien of the venous circuit occurs, accompained by dilatation of vessels, odema in the lungs and an increase in the heart rate.

Etiology:-1-Diseases of the myocardium ,endocardium and pericardium which primarily interfere with the flow of blood into or away from the heart and leading to impairmant of the heart action causing congestive heart failure

2-Vavular diseaseas in case of :

A)-Valvular stenosis or insufficiency.

B)-Rrupture of valve. C)-Congenital valvular defects.

3-Myocardial diseases

A)-Myocarditis [bacterial – viral or parasitic]

B)-Myocardial degeneration. [toxic or nutritional as in case of vitamin E & selenium deficiency]. C)-Congenital and heridetary cardiomyopathy.

4-Pericardial disease:

A)-Pericarditis or hydropeicardium.B)-Pericardial tamponade.

C)-Traumatic pericarditis.

5-Hypertension:-

A)-Pulmonary hypertension. B)-Systemic hypertension

C)-High altitude disease (cattle and buffaloes are highly suciptable to the

high altitude disease because their body adaptation against low oxygen is vey small)

6-Congenital defects.-

A)-Defeets in the myocardium as septal defects. B)-Vascular abnormalities.

Pathogenesis:- 1-When there is overload upon the ejection of the blood from the heart or the contractile power of the myocardium is reduced there are compensatony mechanisms including:-

A)-Increased heart rate. B)-Increased ventricular filling.

C)-Redistribuation of the blood flow.

D)-Cardiac dilation and hypertrophy.

2-These compensatory mechanisms play an important role to maintane the circulatory equilibrium.

But → When these compensatery mechanisms reach their physiological limite and the heart unable to maintain the circulatory equilebrium and circulatory requirement at rest, congestive heart failure develops.

3-Congestive heart failure may occurs in the left ventricle only or in the right ventricle only or both together.

4-Right sided heart failure (Right congestive heart failure)involving the liver, and kidneys causing reduction in their functions (due to heart sided failure manifested only in the greater or larger circulation).

5-Left sided heart failure (Left congestive heart failure)usually restricted to the lesser pulmonany circulation,causing pulmonany odema and pulmonary congestion .

Clinical Symptoms:-

A) In the early stages:

1-The animal easily tiered with respiratory distress.

2-The respiratory rate returne to it's normal rate after excercis after long period.

3-In affected animals there may be evidence of cardiac enlargment and the resting heart rate is moderately increased.

B- Left Side Congestive Heart Failure : [Signs Of Lung Odema].

1)-Increase the respiratory rate and depthe in the rest condition

2)-Moist rales due to pulmonany odema [by auscultation of lower part of the lung].

3)-Bilateral nasal discharge [mucoid discharge] with froothing at the mouth commesure] and the nasal discharge become pureulant if there is secondary bacterial infection.

4)-Moist cough.

5)-Increased the area of cardiac dullnes during percussion of the venteral border of lungs.

6)-Terminally,sever dyspnoea and cyanosis of the mucous membranes.

7)-Tacyhcardia and there may be a murmure referable to left atrio ventricular valve [mitral] or aortic semilunar valve diseases .

8)-Epistaxis in sever cases due to pulmonany congestion and hypertension.

9)-Corneal opacity [glucoma].

C- Right Sided Congestive Heart Failure [Body Odema].

1)-Odema which may be in the form of anasarca, ascites, hydrothorax or hydro pericardium mainly due to hypertension and increase the venous hydrostatic pressure.

2)-Anasarca characterstically to the venteral surface of the body, the neck and the jaw.

3)-Tachycardia and dilatation of the superfacial vien especailly jugular vien.

4)-Feces is normal at first, but in late stage profuse diarrhoea may be present due to involvement of the liver.

5)-Oliguria and albuminuria due to involvement of the kidney.

6)-Right sided congestive heart failure usually concerned with the greater circulation envolving liver and kidney causing disturbances in their function as diarrhoea or abnormal urine.

7)-Body weight may be increased due to odema but the appetite is poor and the conditions is rapidly lost.

8)-Abnormal gait due to general weakness.

9)-Jugular vien pulsation may appeared if the tricuspid valve insufficiency is involved.

10)-Prognosis according to the severity and etiological factors of the disease.

Diagnosis:- 1)-History and clinical symptoms.

2)-Clinical pahtology is if value only to differentiate the causes

3)-Vienepuncture test : increased pressure of blood from needle on vein puncture much than normal due to increasee venous pressure.

4)-Radiography. 5)-ECG. 6)-Echocardiography.

7)-Differential Diagnosis :

A)-***Peritonitis, ruptured bladder, hepatic fibrosis*** :-

*All these conditions cause odema in the abdomen but the heart is normal and usually there is a history of abdominal pain and straining during urination and in case of liver fibrosis there is jaundice and photosensitization.

B)-Normal physiological edema in the late stage of pregnancy, but there is no heart abnormalities and no engorgement of Jugular vein.

C)-Odem also occurs as in hypoproteinaemia as in parasitism (bottle jaw) but it's not severing and usually accompanied by anemia and positive fecal analysis test against the suspected parasites.

8)-P. M. Lesions:- A)-The lesions usually related to the cause and usually concerned with myocardium, pericardium, lung and large blood vessels.

B)-Pulmonary edema and congestion in case of left congestive heart failure

C) - Anasarca – hydrothorax, hydropericardium and engorgement of large blood vessels and enlargement of the liver in case of the right congestive heart failure.

Treatment:- **A*)-Improvement of contractility of the heart*** : By administering cardiac glycoside as digitalis glycosides.

Digitalization.-

1-Digoxin given restrictly interavenous because it is destroyed in the rumen when it given orally, and it not given interamuscular because it causes sever muscular necrosis at the site of administration .

2-Treatment with digoxin leads to:

A)-Improvement of the contractility of the heart.

b)-Decreases the heart rate.

C-Increase the myocardium oxygen consumption.

d)-Increase the cardiac out put.

e)-Decrease the cardiac size i.e. overcome the cardiac hypertrophy.

3-Inicial dose 1.6 – 2.2 mg/ 100 kg Bwt. I.v.

4- Second dose: half of the first dose every 4 hours for 24 hours.

5- Third dose half of the first dose every 12 hours.

***B)-Over come the load of edema*:** By using of diuretic as furesemide (R/ Lasix.) or sodium or potassium citrate.

***C)-Reduce the demands of cardiac output*:** By restriction of activity of the animal.

D)-Vein section:- Can be used as an emergency treatment in case of acute pulmonary edema [4- 8 ml of blood/ kg BWt. may be with drawn].

E)-Paracentesis (aspiration of the fluid from the edema) : from the body cavities.

3- Acute Heart Failure

Definition:-

It's an acute cardiac disorder characterized clinically by:

1-Sudden loss of consciousness. 2-Falling with or with out convulsion.

3-Sever pallor of the mucous membranes and either death or recovery form episode.

Etiology:-

A***)-Sever defect in filling or disorder of filling capacity*** :

As in case of haemepericardium or venous thorobmosis].

B)- Failure of heart as pumps in case of :

1-Myocarditis e.g: encephalomyocarditis virus or FMD.

2-Nutritional deficiency myopathy e.g.: → copper or vitamin E and selenium deficiency

3-Electrocution stricke.

4-Rapid injection of calcium preparations intravenously.

5-Rapid injection of xylazine or concentrated potassium chloride intravenous. 6-Acute anaphylactic shock. 7-Rupture of the aortic valve.

Pathogenesis:- * Mainly reduction in the cardiac out put:

1-All the mentione etiological factors may lead to fall in the minimum volum of the cardiac output causing degree of "tissue anoxia" histotoxic

anoxia

2-The brain is the most sensetive organ which firstly affected ,so the main signe usally in the form of nervous sings.

3-Paller mucosa occurs due to reduction in arterial blood flow.

Clinical Symptoms:-

1-Sudden loss of conciousness due to cerbral anoxia.

2-Staggering and falling with or with out clonic convulsion.

3-Marked paller mucosa.

4-Death usually follow within seconds and usually a ccompained by deep, asphyxial gasps.

5-If there is time for examining the animal there is :

a-Absence of pulse. b-Absence of heart sounds.

c-Bradycardia or tachycardia.

6-In less acute cases the course may be as long as 12-24 hours and dyspnoea and pulmonary odema are prominent signs.

Diagnosis:-1-Hhistory and clinical symptoms.

2-There is no sufficient time for laboratory examination.

Treatment:-Treatment is not usually possible because of the short course of the disease, but the following may be useful.

1-Direct cardiac massage or electrical stimulation but these techniques are restricted to more sophisticated surgical units.

2-Intracardiac injection of very small dose of adrinaline.

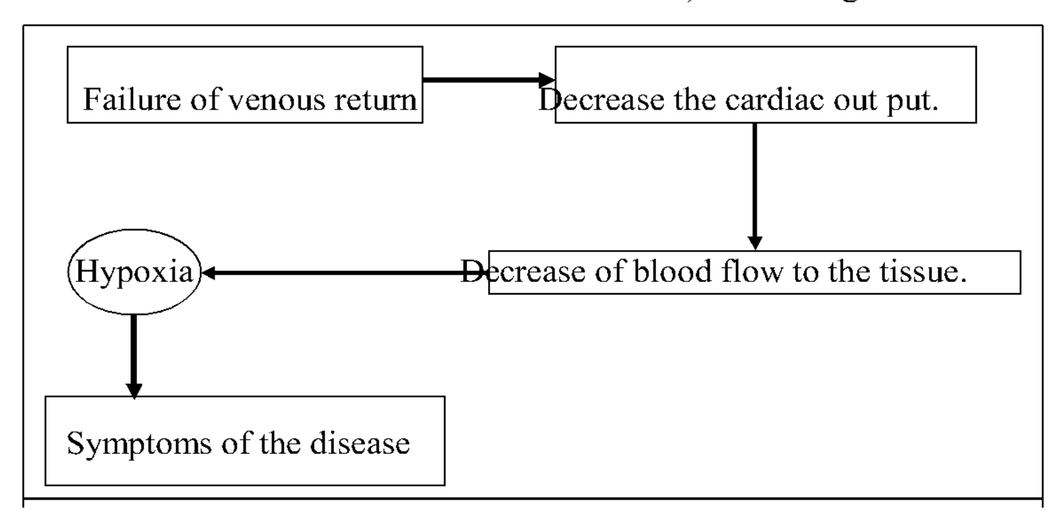
4- Peripheral Circulatory Failure

Definition:-

It's cardiac disorder which characterized by reduction in the cardiac output due to failure in the verous return.

Etiology:-

A)- Failure in the venous return may be: → I)- Vasogenic failure.
II)- Haematogenic failure.



I)-Vasogenic causes of peripheral circulatory failure :

1-occurs when there is sever preipheral vasodilatation

2-In case of shock in which the blood is collected in the splanich vessels. that leading to reduction in the venous return and reduction in the cardiac out put causing tissue hypoxia and reduction in the tissue function.

3-As hypocalcemia.

4-Too sudden reduction in the pressure in the body cavity e.g : by rapid with drawal of ascites fluid or tempany by the trocar and canula .

II)- Haemato genic causes of peripheral circulatory failure

1-Occurs when there is reduction in the circulating blood volume this occurs principally in case of sever loss of body fluids as in cases of

-Haemorrhage -Dehydration -Neonatal calf diarrhoea

Clinical Symptoms:-

1-Anorexia which may accompanied by thirst.

2- Shallow rapid respiration.

3-Sub- normal temperature.

4-Tachycardiac accompanied by weak intensity of heart sounds.

5-Abnormal pulse amplitude.

6-Low arterial blood pressure which can be measured either directly by arterial puncture or indirect method using sphigmography.

7-Nervous signs including :-

-Depression -Listlessness.

-Coma in terminal stage. -Convulsions

8-General weakness with cold skin and extremities .

9-Pale mucous membrane with prolonged capillary filling time(capillary refilling time test).

Diagnosis:- 1-History and clinical symptoms.

2-Laboratory diagnosis **:**

==

A)-PCV, Hb and RBCs count usually below normal due to haemor hagic anaemia and dehydration.

B)-Eosinopenia.

C)-Thrombocytopenia.

E)-Hyperkalaemia, metabelic acidosis and lactic acidosis.

Treatment:-

A)-Regardless of the cause, treatment is to restores the circulating blood volume to normal to avoid tissue anoxia:

B)- Plasma transfusion is required to over come shock.

C)- Give whole blood transfusion to over come haemorrhage. Give isotonic fleuid repracement to overcome dehydration.

D)-Adminestration of large dose of cortico sterodis 2-3mg/kg i.v may be useful as anti cardiogenic shock.

E)-Administerationof sodium bicarbonat 1-2% to overcome acidosis

5- Pericarditis

Definition:- Pericarditis means inflammation of the pericardial sac which resulting in "congestive heart failure" and toxaemia and it may be traumatic (mentioned before) or non traumatic.

Etiology:- 1-localization of blood – born infection in the pericardium.

2-Extension of infection directly from myocarditis or pleurisy.

3-Tuberculosis – streptococcus spp. Infection.

4-Idiopathic fibrinous pericarditis.

5-It may be sequalae of traumatic reticuloperitenitis.

Clinical Symptoms:-

1-The animal reluctant to move .

2- Arched back.

3- Pain on palpation or percussion over the cardiac area of the chest wall inbetween the 3rd. and 6th. intercostal space on the left side beneath and slightly dorsal of the elbow joint.

4-On Auscultation there is frictional or rubbing sound of the cardiac area [due to hyperaemia or congestion of the pericardial sac and deposition of the fibrinous exudate].

5-Temperature may be slightly elevated in the early stage.

6-Abduction of elbow.

7- Shallow abdominal respiration.

8- In advanced cases there is increase in the area of cardiac dullness on percussion over the cardiac area due to exudation of the pericardial sac.

9-If there is gas from gas forming microorganism is present we found that splashing or tinkling sound present on each cardiac cycle.

10-Signs of right sided congestive heart failure (*Odema,tachycardia and engorgement of superficial veins specially jagular vein)

11-Signs of sever toxaemia which depend upon the type of bacteria which present.

12-The animal die usually within 1-3 weeks from congestive heart failure or from toxaemia.

13-The heart sound become less muffled and fluid sounds disappear or persiste in restricted area because of resorbtion of the fluid in the stage of recovery. 14-Complete recovery is not common.

15-Signs of pleurisy may be present as a complication .

Diagnosis:- 1)-History and clinical symptoms.

2)-Laboratory diagnosis : marked leukocytosis and other parameters simillar to that of the traumatic pericarditis

Treatment:- **(See traumatic pericarditis)**

1-If possible, antibacterial treatment for the specific cause of infection or give broad spectrum antibiotics or sulphonamide as a non specific treatment.

2-Repeated paracentesis to relieve the fluid pressure in the pericardial sac but this give only a temporary relief because the fluid returing quickly.

3-Diuretics is recommended, but digitalization is usually not very effective and is dangerous if infection stell present. 4-Surgical treatment.

6- Venous Thrombosis

Definition:- Venous thrombosis means development of thrombi in the veins and may result in : → Local obstruction to venous drainage or Liberation of emboli which may lodge in the brain, lungs liver or other vital organs.

Etiology:- **P**helebitis is the common origin of thrombi which may be caused by: 1-Infection of the umbilical vein in the neonatal calves

2-Extension of infection from surrounding diseased tissues

3- Localization of blood born infection eg: bacteraemia or septicemia.

4- Injection of irritant or semisolid materials in the major vien .

Clinical Signs:- 1-Engorgement of the vien. 2-Local odema.

3-Pain on palapation of the area drained by the affected vien.

4-Rupture of the vien may occurred causing extermal or internal fatal haemorrhage.

N.B → odema of perineum, udder and ventral abdominal wall may occurs due to pressure (not- phlepitis and thromabosis) of faetus during pregnancy.

Treatment:- 1-Parenteral anti bacterial drugs to overcome phlebitis and prevent the secondary bacterial infection .

2-Hot fomentations to external veins to overcom the obstruction of the vein and subsided the swelling.

3-Adminsterationof diuretices to reliefe the edema.

7-Myocardial Diseases & Cardiomyopathy

Etiology:-

A number of diseases are accompanied by inflammation, necrosis or degeneration of the myocardium. These diseases may be viral, bacterial parasitic or nutritional deficiency.

* The term cardiomyopathy is restricted to the diseases which characterized mainly by myocardial damage.

* causes of the myocardial diseases may be :

A- Bacterial myocarditis:-

1- Streptococcuss spp infection. 2- T. B.

3- Clostredium chauvoei (Black leg disease).

4- Following bacteraemia 5- Extension from peri- or endocarditis.

B- Viral myocarditis:-

1- F.M.D in young calves. 2- Viral leucosis in cattle.

3- Reft vally fever (virus).

C- Nutritional deficiency:-

1- Vite E/selenium deficiency.

2- Chronic copper deficiency in cattle [falling disease].

3- Cobalte deficiency. 4- Iorn deficiency.

D- Poisoning causes:-1- Selenium, arsenic, mercury, phosphorus and

thalium poisoning.

2- Drugs : as catecholamine preparations which lead to multifocal myocardial necrosis specially in the left ventricle.

3- Vit- D toxicity [causes calcification of the heart muscules], salinomycin, or monensin toxicity.

E- Myocardial disease may be heridetary.

F- Myocardial diseases may be asequala of congestive heart failure due to the effect of congestive heart failure on the myocardium and the function of the heart as a pump.

Clinical Symptoms :-

A- In Early stage or milde or moderate myocardial damage stage :

1- Decreasing the activities of the animal.

2- Increase the heart rate and size but it may be not detactable.

3- Tacharrythmia with multiple ventricular extrosystoles.

4- Changes in the pulse and heart sounds.

5- The signs may not appeared at rest but become clear after exercise or excitement due to the effect of catecholamine (adrenaline & noradrenaline on the affected myocardial muscules).

6- Temperature may be elevated if the causes are infectious (bacterial or viral).

B- In late stage or stager of more sever myocardial damage :

1- Sudden death or attack of cardiac syncope due to heart failure (acute).

2- Sever dyspnoea or general odema due to congestive heart failure.

3- Myocardial weakness is frequently accompanied by systolic murmures

N. B:

A)-Signs of arrhythmia and congestive heart failure usually accompanied by myocardial diseases.

B)-Other symptoms of the etiological diseases of myocardial diseases also appeared as in case of FMD.infection.

Diagnosis:-

A- History and clinical symptoms.

B- Laboratory diagnosis :

1- Mycordial damage or necrosis leads to release and increase the level of the cellular enzymes as → Aspartate transaminase AST(GOT).

→ Lactate dehydrogenase.

→ Creatine phosphokinase CPK.

C)-Microbiological parasitological and toxological examination to conferme the suspected causes.

D)-Examination of the nutritional trace elements defeciency.

E)-PM. Lesions:-

1- ***In bacterial myocarditis***:-

Usually accompanied by abscessiation or small areas of inflammation in the myocardial muscles.

2- In viral myocarditis, nutritional or poisoning causes :

Presence of pallor streaks inbetween the apparantly healthy

muscular bundles as in case of distrofic nutritional muscular disease (Vitamin E and Selenium deficiency).

3- In acute form :

*Linear or peticheal haemorrhage may be present on the myocardial muscules.

4- Calcification of the myocardial muscles as in Vit- D toxicity.

5- In late stage the myocardial muscles may be replaced by fibrous tissues.

6- The heart is flabby, thin walled and show patches of shrunken and tough fibrous tissues.

7- Atrial rupture may be present as in case of sudden death.

F)-Differential diagnosis:-

1)-Pericarditis: Characterized by:-

-Frictional sound in early stage.

-Muffled sound in late stage.

-Jugular vein pulsation.

-Positive pain test.

2)-Other diseases which may lead to congestive heart failure.

Treatment:-

****Mainly directed to the main etiological factors.***

8-Endocarditis

Definition:- Endocarditis means that inflammation of the endocardium with interference with the ejection of the blood from the heart causing insufficiency or stenosis of the valve with resultant cardiac insufficiency or heart failure.

Etiology:- Usually bacterial infection as :

1-Clostridium chavoei (Black leg disease). 2-Erythseplothrix infection.

3-Conynebecertium or actinomyces infection.

4-Myocoplasma infection. 5-Alpha. Hemolytic streptococcus.

Clinical Symptoms.-

1-History of ill– thrift. 2- Poor conditioned animal.

3- Temporary fall in milk production. 4- Persistent tachycardia.

5- Murmurs on auscultation according to the affected valve (see the valvular diseases)

6- At all stages a moderate fluctuating fever is common with affection of other organs of the body due to migration of bacterial emboli through the blood vessels and localized in any parts of the body leading to either.

-Embolic pneumonia. -Arthritis (lameness). –Nephritis

-Tenosynovitis. -Peripheral lymphadenitis or myocarditis.

7- Increase the pulse rate with pale mucosa.

8- Thrilling on palpation of cardiac area.

9-Sings of congestive heart failure.

10- The course is very long (several weeks to several months) or the animal may drop dead without previous illness.

11- Decrease the activities of the animal.

12- Acute heart failure may occur.

13- Rupture of the chordae tendinae of the mitrial (bicuspid) valve may occurs as a sequalae of endocarditis which manifested by acute heart failure or left sided congestive heart failure and death.

Diagnosis:- A- History and clinical symptoms.

B- Clinical pathology: → Neutronphilia – leukocytosis. -Monocytosis
-Anemia. -Increase the plasma level of fibrinogen.
- Hypergamaglobulinemia is characterized of chronic bacterial infection.

Treatment:-A- Usually expensive and is not highly successful due to:

1- Difficulty of controlling the infection or to detect the sensitivity test.

2- The thickness of the lesion prevents adequate penetration of the drug to the endocardium.

So: The antimicrobial drugs should be taken in massive dose to rise it's concentration in the blood.

B- Isolation of the animal and use of Penicillin with Gentamycin or use of potential high dose of sulphonamide.

C- The response of treatment is evident by falling in body temperature.

D- Treatment required for very long period up to 4 months.

E- If congestive heart failure is present the prognosis is poor.

9-Vavular Diseases

Definition:- It's a group of diseases of the heart valves which interfere with the normal flow of the blood through the cardiac orifice resulting in murmurs, cardiac insufficiency or in sever cases congestive heart failure.

Etiology:-

1-Acute or chronic endocarditis is the more common form.

2-Laceration, detachment of valves or rupture of chordae tendinae occurs much less commonly but can occurs spontaneously or secondary to endocarditis.

3-Congenital valvular insufficiency.

4-Chronic trauma of the valve leaflets was considered important initiating factors.

5-Excessive dilatation of the atrioventricular valve such as occurs in the Brisket disease [is sporadic disease of cattle and possibly other species which kept at high altitude, clinically characterized by congestive heart failure] and secondary to the myocardial disease may result in functional insufficiency of the valves.

6-C.H.F(congestive heart failure).

N.B: → Valvular stenosis: means imperfect opening of the valves.

→ Valvular insufficiency: means imperfect closure of the valves.

Clinical Symptoms:-

1-Signs of congestive heart failure.

2-Murmurs which may be:

A-Systolic murmurs: Which occurs with the systolic heart sound due to insufficiency or stenosis of the atrioventricular valves and this sound occurs with the systolic (lupp) sound or in between the systolic and diastolic sound.

B-Diastolic murmurs: which occurs due to stenosis or in sufficiency in the aortic or pulmonary semi lunar valves, this murmurs occurs commences with the diastolic phase or in-between the diastolic phase and the next systolic phase.

3-According to the degree or intensity and loudness of the murmurs it may be the following Grade.

A-Grade I: the faintest audible murmur, generally only detected after careful auscultation.

B-Grade II: a faint murmur that is clearly heard after only a few seconds auscultation.

C-Grade III: A murmur which is immediately audible as soon as auscultation begins and is heard over a reasonably large area.

D-Grade IV: An extremely loud murmur accompanied by a thrill. The murmur becomes in audible if the stethoscope is held with only light pressure on the chest.

E-Grade V: An extremely loud murmur accompanied by thrill, the murmur can still be heard when the stethoscope is held with only light pressure against the chest.

4- Grade I is not clinically significant.

*Grade V and IV are invariably.

*Grade II and III are significant according to their cause.

The forms of valvular disease may be:-

1- Stenosis of the aortic valve. 2- Insufficiency in the aortic valve.

3- Stenosis and in sufficiency of pulmonary valve [rare].

4- Insufficiency of the left atrioventricular (mitral) valve.

5- Insufficiency of the right atrioventricular (tricuspid) "more in cattle & sheep". 6- Stenosis of right or left atrioventricular valve.

* ***Location of different valve at which the abnormal sounds can be heard by auscultationare :-***

1- Right atrioventricular valve: Opposite to 4^{th} rib about 10 cm. from the costochondral junction (dorsally).

2- Left atrioventricular valve: Mainly opposite to 5^{th}. Inter costal space.

3- Pulmonary semilunar valve: Opposite to 3^{rd}. inter costal space opposite to olecranon process of the ulna.

4- Aortic semilunar valve: Opposite to 3^{rd}. rib about 12cm. above the sternum.

Diagnosis:- 1- History and clinical symptoms.

2- Clinical pathology according to the specific cause.

Treatment:-

1-There is no – specific treatment of the valvular disease.

2-Treatment of congestive heart failure of endocarditis can be used.

Chapter No(3)

Respiratory System

1-Pulmonary congestion & Edema
2-Pulmonary Emphysema
3-Pneumonia
4-Pulmonary Abscess
5-Calf Diphtheria
6-Paranasal Sinusitis
7-Rhinitis

1-Pulmonary Congestion & Edema

Definition:- Pulmonary congestion means that increase in the amount of blood in the lungs due to engorgement of the pulmonary vascular bed, the pulmonary congestion usually followed by pulmonary edema due to escaping of the intravascular fluid into the parenchyma .

Etiology:-

Pulmonary congestion may be (primary congestion – secondary congestion).

A)-Primary pulmonary congestion as in case of :

1-Early stages of most cases of pneumonia.

2-Inhalation of smokes and fumes.

3-Anaphylactic reaction.

4-As a complication in recumbent animals.

B)-Secondary pulmonary congestion as in case of :

1-Congestive heart failure.

2-Pulmonary edema usually occurs as squeal of pulmonary congestion as in cases of:

-Acute anaphylaxis.

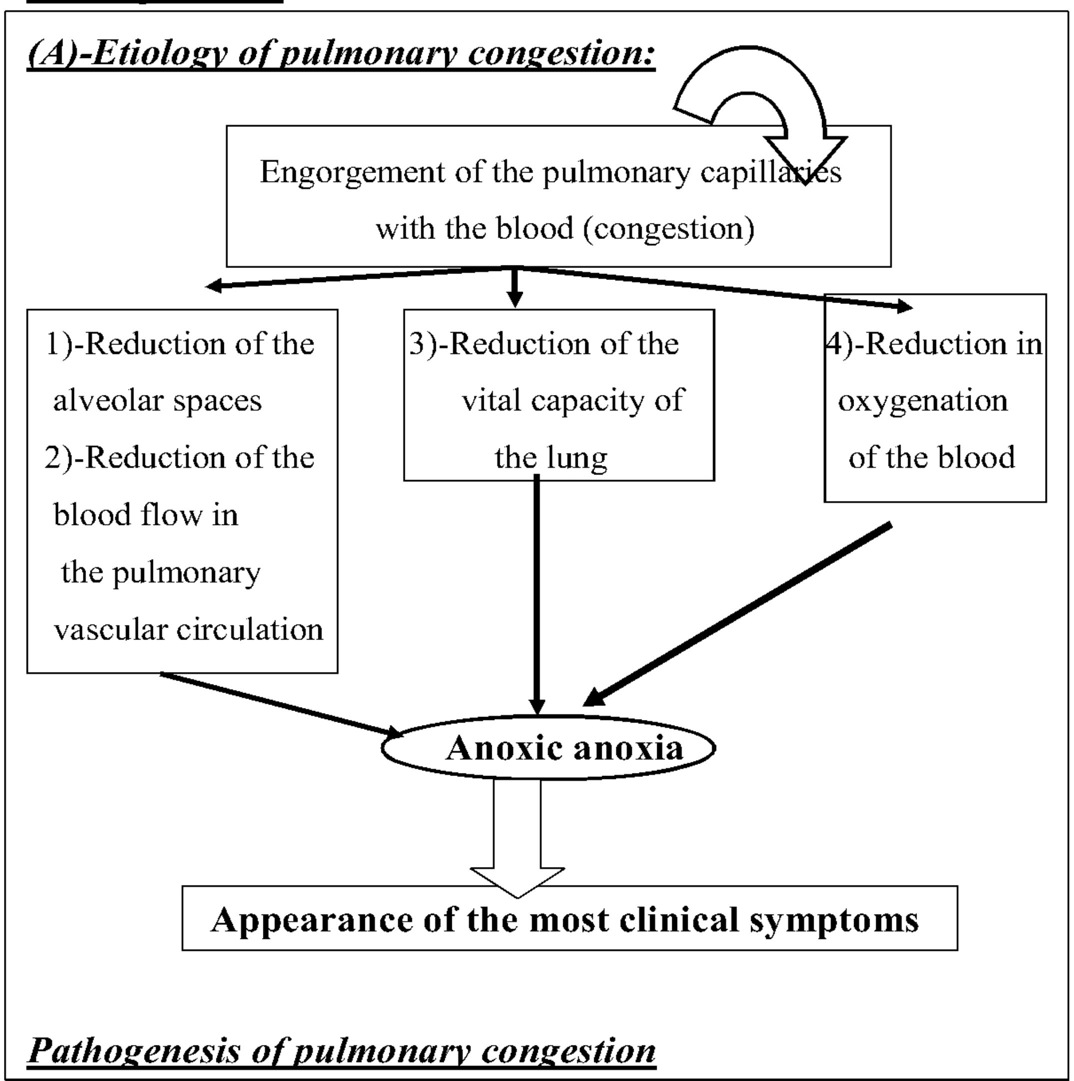
-Acute interstitial pneumonia.

-Poisoning with organophosphorus.

-Myocardial form of .vitamin E and selenium deficiency

-Pulmonary or alveolar damage or injures.

Pathogenesis:



Pathogenesis of pulmonary congestion

(B)-Anoxia also leads to increase the pulmonary vascular permeability and increase the venous hydrostatic pressure in the capillaries which causing edema (second stage of congestion).

Clinical Symptoms:-

1-Increase the respiratory depth with dyspnoea, mouth breathing and dilated nostrils [signs of dysponea].

2-There is characteristic or marked abdominal and thoracic movement during expiration and inspiration.

3-Extension of the neck.

4-The head is hung low.

5-Increase the respiratory rate specially if there is hyperthermia as in case of pneumonia, anaphylaxis or violent exercise.

6-Abduction of the fore limbs.

7-The pulse rate is elevated up to 100/min.

8-Bright red nasal mucosa in the first stage then becomes cyanotic due to reduction in the oxygenation of the blood [Anoxia].

N.B.:

*Pulmonary congestion may be associated with pulmonary hypertension and increase the resistance of pulmonary circulation leading to ventricular hypertrophy and dilation ended by congestive heart failure.

9-Soft and moist cough.

10-Serous nasal discharge in the stage of congestion but in case of edema it become voluminous, frothy nasal discharge which may be blood tinged.

11- Auscultation may reveal rnoist rales in the ventral part of lungs.

12-Percussion varies from normal resonant in congestion to dullness

in case of pulmonary edema.

13-Stage of pulmonary congestion usually is an indicator to lungs or heart disease.

14-Spontaneous recovery may occur but pulmonary edema usually indicates to a stage of irreversibility specially if there is damage to alveolar epithelium, or myocardial affection.

15- Death in case of pulmonary edema usually due to respiratory failure.

Diagnosis:-

A)-History and clinical symptoms.

B)-PM. Lesions:-

1-The lungs are dark red in color.

2-Excessive quantities of venous blood exude from the cut surface.

3-Swelling and loss of elasticity with pit on pressure in case of edema and the color is become paler than normal.

C)-Histological examination:-

-Damage of pulmonary capillaries

-Pulmonary capillaries are engorgement with blood and transudation -Hemorrhage are evident.

Treatment:-

1-Correction of the primary causes [pneumonia, vitamin E and selenium deficiency ….etc].

2-The animal should be kept in rest, clean and good ventilated environment.

3-Epinephrine (adrenaline) is recommended in case of pulmonary edema due to anaphylaxis.

4-Corticosteroide which (maintain the vascular integrity and decrease the pulmonary vascular permeability and decrease the occurrence of pulmonary edema).

5-Antihistamenic is recommended with epinephrine in anaphylaxis but acetylsalicylic acid (aspirin) is more effective than antihistaminic.

6-Slow intravenous administration of Aminophylline will help the dilating bronchioles [it acts as bronchodilators].

7-Removing the fluid from the lung by intravenous administration of quick acting diuretic as Furosemide (Lasix).

8-If pulmonary edema is due to organophosphorus poisoning administration of atropine sulphate is recommended.

2-Pulmonary Emphysema

Definition:-

Pulmonary emphysema means distension of the lung caused by over distension of alveoli with rupture of alveolar walls with or without escape of air into the interstitial spaces. Characterized be dyspnoea, forced expiration, poor tolerance and hyperpnoea.

Etiology:-

1-Acute interstitial pneumonia.

2-Bronchiolitis.

3-Secondary to bronchopneumonia.

4-Perforation of the lung by foreign body as in case of traumatic reaticuloperitonitis.

5-Acute chemical injury as in inhabitation of harmful fumes.

6-Pulmonary abscess.

7-Chlorine gas poisoning.

8-Parasitic pneumonia with the pulmonary edema

Pathogenesis:-

1-Pulmonary emphysema result mainly due to weakness or loss of the elasticity of the alveolar wall which lead to accumulation of air in the alveoli → (over-distension of the alveolar wall and finally ended by rupture of the alveoli).

2-Accumulation of gases in the alveoli including carbon dioxide leads to:- A)-Stimulation of respiration and increase the death of respiration.

B)-Acidosis and anoxia.

C)-Disturbances in the metabolism in the body.

D)-Decrease the negative pressure in the lungs which may lead to right ventricular failure specially if there is primary defect in the myocardium.

Clinical Symptoms:-

It's usually undetectable but it may be characterized by :

1-Sever dyspnoea (expiratory dyspnoea).

2-Forced expiration with grunting on expiration.

3-Loud crackling (rales) on auscultation.

4-Usually associated with pulmonary edema (due to disturbance in the pressure balance i.e. hydrostatic pressure of the capillaries and plasma osmotic pressure.].

5-Intermittent bilateral nasal discharge.

6-Normal animal body temperature and appetite.

7-Pulse rate may be increased due to pulmonary arterial hypertension.

Diagnosis:-

1-History of the case and clinical symptoms.

2-Laboratory diagnosis (there is non-specific hematological picture).

a- Acidosis due to Co_2 retention.

b- Polycthemia as a compensatory action against anoxia.

3-Differential diagnosis:-

a- Pneumonia (usually accompanied by fever - localized abnormal sounds by auscultation.

b- Pneumothorax (usually accompanied by forced inspiration – Absence of normal breath sounds).

Treatment:-

-There is no-specific treatment of pulmonary emphysema but suing of:

A) Corticosteroids.(Dexamethason-Phenylojector Megloxin)

B) Antihistaminic.(Avil or Vetibenzamine)

C) Administration of oxygen in valuable animal.

D) Correction the primary causes may give good result in treatment of pulmonary emphysema.

E) Bronchodilators. As: 1-Parasympatholytic bronchodilators as:

*Atropine sulphat. IV 0.02 mg/kg Bwt..(1cm./20-200 kg.Bwt. IM. Or S/C).

2- Aminophyllin: xanthine derivatives. Acts directly on the smooth muscle fibers

3-Pneumonia

Definition:-

Pneumonia means inflammation of the lung parenchyma usually accompanied by inflammation of the bronchioles (broncho-pneumonia) and often inflammation of the pleura (pleuropneumonia) clinically characterized by dyspnoea, cough and abnormal respiratory sounds on auscultation.

Etiology:-

A)***Predisposing factors :***

1- Sudden change in the weather.

2- Poor ventilated housing which leads to accumulation of ammonia.

3- Stresses of transportation or weaning or over crowding.

4- Inhalation of irritant gases or smokes.

B) Infectious causes:

1- Viral infection as:-

- Para influenza-3. - Reovirus - Bovine herpes virus 1 [IBR].
- Bovine respiratory syncytial virus.
- Viral interstitial pneumonia: in the recently weaned beef calves caused by bovine syncytial resp. virus.

2-Bacterial infection:-

-Chlamydia spp. -Actinobacillus spp.

-Mycoplasma spp. [contagious bovine pleuropneumonia caused by mycoplasma mycoides].

-Mycobacterium bovis [pneumonia tuberculosis].

-Fusebacterium necrophorus pneumonia [sporadically and accompanied by calf diphtheria].

-Pneumonic pasteurellosis (shipping fever) caused by pasteurella multocida or haemolytica with or without parainfluenza -3 virus.

-Actinomyces (corynebacterium pyogenes).

3-Parasitic pneumonia:

(Dictyoculas viviparous - migrating larvae of Ascarasis.

4-Mycotic may be:

as Asperagellosis (klebsiella pneumonia in calves).

Clinical Symptoms:-

1-Rapid and shallow respiration is the cardinal signs of early pneumonia.

2-Dyspnoea occurs in later stage when much of lung tissue is non functional.

3-Cough which may be :-

*Dry frequent cough in interstitial pneumonia.

*Moist, painful cough in bronchopneumonia.

4-Cyanosis: Not a common sign, occurs only when large areas of the lung are affected.

5-Nasal discharge may or may not present depending upon the amount of exudates present in bronchioles and whether or not there is accompanying inflammation of the upper respiratory tract.

6-Abnormal odor of the breath which may be :-

*Decay: when there is a large accumulation of pus.

*Putrid: when there is lung gangrene.

7-The pulse rate increased due to pulmonary arterial hypertension.

8-Toxemia, fever, anorexia and depression in acute bacterial bronchopneumonia.

9-Death usually occurs due to pleurisy, gangrenous pneumonia, asphyxia or due to heart failure or respiratory failure.

*Bacterial pneumonia → i.e. presence of exudates→ presence of moist rales

*Viral pneumonia → i.e. absence of exudates → presence of dry rales.

10-Auscultation of the lung varies according to the stage of pneumonia.

A-In the early stage of congestive stage: of bronchopneumonia and interstitial pneumonia there is increased bronchial sound [breath sound].

B-Crackles (moist rales) develop in bronchopneumonia as exudation increases.

C-Clear, harsh bronchial sounds are audible in uncomplicated interstitial pneumonia.

D-Loud bronchial sound occurs when complete consolidation is occurs (consolidation also causes increased audibility of heart sounds).

E-Frictional sound may occurs if pleurisy is complicated with pneumonia (plueropneumonia).

F-Muffling of bronchial sounds occurs in the late exudative stages i.e. gray hepatization.

11-Course of the disease is about: 1-3 weeks.

Complications of pneumonia:-

1-Pleurisy due to extension of the infection causing pleuropneumonia.

2-Pulmonary abscesses.

3-Acidosis due to Co_2 retention.

4-Ventricular hypertrophy and heart failure may occurs

5-Gangrenous pneumonia.

6-Pneumothorax.

N.B.:

Cattle are highly susceptible to be infected by pneumonia that due to it has normally low physiological capacity of the gaseous exchange. So if it exposed to physical or metabolic activity or present in high altitude or in bad ventilated house it lead to low oxygen level in the bronchioles and alveoli causing hypoxia which reflection on reduction of the mucocillary defense mechanism and predispose of pneumonia.

Diagnosis:- 1-History and clinical symptoms

2-Nasal swap and endoscopic examination

3-Bacteriological, parasitological or serological analysis to detect the main causative agent

Treatment:- 1-Isolation of the affected animals especially if infectious diseases are suspected.

2-Isolation must be in warm, well ventilated, draft free place and provided by fresh water and light.

3-Nourishing food is required and parental feeding if the animal not eat (i.e. anorexia).

4-The choice of antibacterial agent [antibiotics or sulphonamide] based mainly on the culture and sensitivity test or choice of anti-parasitic agent if verminous pneumonia is suspected.

5-The use of corticosteroid as dexamethazone as an anti-inflammatory as it → maintain the integrity of the pulmonary vascular circulation and decrease the permeability of the pulmonary vessels, so it help in avoid of occurrence of pulmonary edema.

6-The use of anti histaminic as Avil. The use of bronchodilators as Aminophyllins and Theophylline .

7-The use of expectorants according to the type of cough:-

A-Painful, exhausting cough and secretion is tenacious → we can use sedative expectorants such as: ammonium or potassium salt which stimulate secretion of protective mucous and lessen coughing.

B-Soft cough and the bronchial exudates is voluminous as in chronic bronchopneumonia the stimulant expectorant is more valuable.

C-Exhausting cough and interferes with activity but there is little exudation the anodyne expectorant is recommended as Belladona, Codine, Morphine.

4-Pulmonary Abscess

Definition:-

Its development of single or multiple abscesses in the lung causing a syndrome of chronic toxemia, emaciation, coughing and followed by supurative bronchopneumonia.

Etiology:-

A)-Primary causes:-

1-Tuberculosis.

2-Actinomycosis.

3-Asperagellosis and histoplasmosis.

4-Corynebacterium.

B)-Secondary causes:-

1-Bovine pleuropneumonia.

2-Aspiration pneumonia

3-Emboli from [endocarditis, mastitis, metritis or omphalophlepitis].

4-Penetrating foreign body as in case of traumatic reticuloperitiuritis.

Clinical Symptoms:-

1-Signs of toxemia [dullness, anorexia emaciation and fall in milk production].

2-Temperature usually elevated and fluctuated.

3-Short, harsh and painless cough.

4-Intermittent episodes of epistaxis or hemoptysis which terminated in fatal pulmonary hemorrhage following erosion of the adjacent large pulmonary vessels which present near to the pulmonary abscess.

5-Dullness on percussion or auscultation over the area of abscess.

6-Feted odor of the nasal discharge which indicate to development of bronchopneumonia from the extension of the abscess.

7-The respiratory distress varies according to size and location of the abscess..

Diagnosis:-

1-History and clinically symptoms.

2-Nasal swap and radiography.

3-Hydatid cyst and pulmonary tumor give the same symptoms but they are not accompanied by signs of toxemia.

Treatment:-

1-Usually not successful.

2-Slaughter of the animal.

3-If the causes are corynebacterium we can give Erythromycin 25mg/kg Bwt. 3 times daily and Refampicin 5mg/twice daily for 4-weeks [expensive].

5-Calf Diphtheria

Definition:- It's a disease of calves affecting the oral cavity and larynx causing necrotic stomatitis or necrotic laryngitis and characterized by high fever and swelling and ulceration of the affected structures.

Etiology:-

* Fusebacterium necrophorum infection preceded by traumatic injury of mucous membranes of oral cavity or larynx by coarse feed or feed containing an excessive quantity of tough stems as a predisposing factor.

Clinical Symptoms :

A)-Calf diphtheria usually occurs as necrotic stomatitis in calves less than 3 months of age and as necrotic laryngitis in older calves.

B)-The calf with necrotic stomatitis has:

1- Difficulty in nursing.

2-Temperature may rise up to 40°C.

3- Depressed appetite.

C)-The calf with necrotic laryngitis has : (check abscess is characteristic lesion in the calves)

1-Load wheezing (most prominent sign in sever cases).

2-Temperature may rise up to 41°C.

3-Salivation and protrusion of the tongue.

4-Nasal discharge and rapid respiration.

5-Cough as the lungs often become involved.

6-Dehydration and emaciation.

7-Untreated calves suffering from pneumonia and toxemia within 2-7 days.

Treatment:-

1-Isolation of the affected animals.

2-Antibiotics and sulphonamides are recommended.

A-Antibiotics :

1- The antibiotics of choice are: penicillin [procaine penicillin suspension 5,000-10,000Iu/L.b.I/M].

2- Penicillin and streptomycin [5-10 mg/lb dihydrostroptomycin aqueous suspension I/M].

3- Chloramphenicol (5-10 mg/Lb 3 times/day.)

B- Sulfonamide:

- Sulfamerazine sodium 130mg/kg but followed by 65 mg/kg daily at least 4 days.

- Sulfamethozine sodium orally or preferably I.V. 130 mg/kg Bwt. followed by 65 mg/kg orally daily for at least 4 days.

1- Surgical interference in case of check abscess.

6-Paranasal Sinusitis

1-There are more than pairs of the Para nasal sinuses but there are main two sinuses of clinical important.

2-Maxillary and frontal sinuses (maxillary sinuses present in front of the zygomatic bone while the frontal sinuses present above the imagine line that connected the two eyes).

3-Paranasal sinuses can be examined clinically either by :-

A)-Inspection:-

To detect any abnormal swelling or enlargement in the area of the sinuses

B)-Palpation:-

To detect the nature of the abnormal swelling either it is inflammatory (hot and painful palpation) or non inflammatory (cold and painless palpation).

C)-Percussion:-

The normal percussion of the Para nasal sinuses is tympanic sound.

D)-Sinocsope

Also can be used in the examination of the Para nasal sinuses

4-Para nasal sinusitis means that inflammation of the Para nasal sinuses which may be uni-or bilateral

Etiology:-

1-Trauma

2-Dehorning act as a predisposing factor of the para nasal sinusitis specially when it is occurred under aseptic condition.

3-Extension of the infection from the upper respiratory tract.

Clinical Symptoms:-

1-Shaking of the head

2-Body temperature remain normal until there are any systemic disturbances

3-Uni or bilateral nasal discharges which is firstly serous and it become purulent due to secondary bacterial infection.

4-Abnormal respiratory sound.

5-Painful palpation over the affected sinus.

6-Percussion revealed dullness sound due to the air of the sinuses was replaced by exudates.

7-The nasal discharge occurred spontaneously when the animal lowering the head.

8-Painful mastication.

9-Inclination of the head toward the affected side.

10-Paranasal sinusitis may lead to dental fistula.

Diagnosis:-

1-Case history and clinical sings

2-Palpation and percussion

3-X-ray or sinuscopy

Treatment :-

1-Hot fomentation over the skin of the affected sinus

2-Broad spectrum antibiotics (Oxytetracyclin,Streptoomycin or Penicillin)

3-Analgesic to relief the pain (Analgin, Novalgin or Aspirin)

4-Anti-inflammatory (Megloxin, Pheneloject or Dexamethazon)

5-Surgical interference by what so called trephination

7-Rhinitis

Definition:-

Rhinitis means inflammation of nasal mucous membrane characterized by sneezing, wheezing and abnormal sound during inspiration as well as nasal discharge which may be serous, mucoid or mucopeurelant according to etiology.

Etiology:-

1-Contagious ecthyma (rare)
2-Allergic rhinitis.
3-IBR(Infectious Bovine Rhinotraciaitis)
4-Melodiosis

*Atopic rhinits (means that defects in immuno system causing allergy as rhinits).

Pathogenesis:-

1-Rhinitis is of minor importance except when it lead to obstruction of the nasal cavity leads to difficult in respiration.

2-Inflammed nasal mucous membrane may be erroded and may help in secondary bacterial invasion.

3-Rhinitie itself not considered as a disease but it may considered as a complication of other diseases as pox.contagiuos eczyma …………etc.

Clinical Symptoms:-

1-The cardinal (main) signe of rhinitis is nasal discharge which is serous firstly and become mucoied and after bacterial invasion it become peurulent.

2-Erythema, errosion or ulceration may be observed by inspection of the nasal mucosa.

3-Sneezing followed by snorting sound.

4-The disease may unilateral or bilateral.

5-Mouth breath in case of bilateral affection.

6-The animal may rub it's nose along the ground due to irritation.

7-Symptoms of specific disease as pox, nasal myiosis,IBR……etc.

Diagnosis:-

1-Clinical signs and case history.

2-By using of flexible fibrooptic endoscope or rigid endoscope.

3-Observation of larvae in case of oestrus ovis infection.

4-Differential diagnosis from inflammation of the facial sinuses in which the nasal discharge is continious and persist and there is no irritation.

Treatment:-

1-Treatment the specific disease.

2-Removing the tanacious excudate which causing obstruction of the nasal cavity and flushing the nasal cavity by saline solution or worm water.

3-Nasal decongestant sprayed up into the nostrils may provide some relief

R/vibroseal R/prosoline.

4-Anti-histaminic preparations specially in allergic rhinitis.

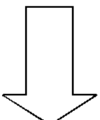
R/ Avil R/Allecure R/ Tavegyl R/Vegebenzamine.

5-Systemic antibiotics to prevent the secondary bacterial invasion.

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Chapter No.(4)

Urinary System



1- **Renal Ischemia**
2- **Pylonephritis**
3- **Renal abscess or emboli**
4- **Cystitis and urolithiasis**

1- Renal Ischemia

Definition:-

Renal ischemia is a pathological condition attributed to acute or chronic reduction in the blood flow through the kidney and characterized by transitory oliguria followed by anuria and uremia.

Etiology:-

1-Usually caused by general circulatory diseases e.g. shock, dehydration.
2-Acute hemorrhagic anemia and acute heart failure.
3-Usually caused by chronic circulatory insufficiency e.g. congestive heart failure (right) which leads to decrease the renal blood flow causing reduction in the glomerular infiltration and oliguria or anuria.

Pathogenesis:-

A) Reduction in the renal blood flow leads to reduction in the glomerular filtration and subsequently the following:
1- Elevation of the level of normally excreted metabolites in the blood stream e.g.: blood urea nitrogen (BUN) causing pre-renal uremia as in case of congestive heart failure.
2- Oliguira or even anuria and uremia.
B- Reduction in the blood flow if sever and persisted for long period the resulted reduction in the blood flow usually followed by : anoxic degenerations in the renal parenchyma [Nephrosis]which varies from tubular necrosis to cortical necrosis.

Clinical Symptoms:-

1-Renal ischemia usually doesn't appear as a disease entity due to it's masked by the signs of other primary causes as shock, heart failure ….etc.

2-Oliguria or even anuria associated with the picture of uremia as anorexia, emaciation, stomatitis, encephalopathy………….etc.

3-The only observed clinical signs of renal ischemia may be the clinical picture of uremia associated with oliguria or anuria.

Diagnosis:-

1-History and clinical symptoms.

2-Laboratory diagnosis : A) High level of BUN. B) Protenuria

N.B. Evidence of oliguira and uremia together with heart failure suggests renal ischemia.

Treatment:-

1-Correction of the circulatory disturbance or other etiological factors.

2-Supportive treatment as fluid therapy to compensate the loss in the body fluid.

2-Pyelonephritis

Definition:--Pylonephritis is a sub, acute or chronic, supurative inflammation of the kidney and the pelvic portion of the kidney, it's highly fatal specific disease of the cattle caused by corynebacterium renal characterized by chronic purulent inflammation of the kidney, ureters and bladder. -It affect the adults only in sporadic cases

Etiology:- 1-Mainnly corynebacterium renal, but it may be considered as mixed infection due to there are other bacteria associated with corynebacterium renal as streptococci.

2-Renal abscess may be acts as predisposing factor of pyelonephritis.

Incidence And Occurrence:-

1-The disease most common in the form of sporadic cases

2-High mortality rate with high economic loss especially in non-treated cases

3-Most common in cow and most in female than male

4-It is considered as a stress related diseases as stress due to cold weather and high protein in the ration act as a predisposing factor of the disease

5-Urehteral catheterization or other instrument may play a role in the entrance of the infection to the urinary tract.

6- Artificial insemination or matting from the infected bull leads to spreading of the infection in the farm.

7- Infected animal is considered as the main source of the infection.

Pathogenesis **: -**There are many factors which enhance the pathogenesis of the diseases this factors are

1-Infection of the urinary tract should be present.

2-Stagnation of urine which permits the multiplication of the bacteria stagnation of urine may occur as in case of urolithiasis, inflammatory swelling or debris, or pressure by the gravid uterus.

Clinical Symptoms:-

1-The first sign may be passage of smoky or blood stained urine.

2-In other cases the first signs may be attack of acute colic, such attacks due to obstruction of the urethra, and renal pelvis by pus or tissue debris.

3-Loss of appetite and condition and decrease in milk yield over a period of week. 4-Fluctuating temperature

5-Sometimes systemic reactions may be present.

6-Frequent painful urination.

7-Pain on pressure of the transverse process of lumber region

***8-Rectal palpation reveals*.-**

A-Thick tender wall of the urinary bladder.

B-One or both kidneys show enlargement and absence of lobulation with pain on palpation.

9- Sings of the abdominal pain as kicking of the abdomen and abduction of the hind legs and arched back

Diagnosis:-**1**-History and clinical finding.

2-Isolation of the causative agent from the urine sample

Treatment :-

1-Large massive dose of penicillin for long duration is the drug of choice 11-22000 iu /kg.Bwt. IM.

2-Acodification of urine.

3-Urinary antiseptic e.g.: Hexamine 15 gm 40% solution I/U after acidification of urine.

4-Overcomming the poor condition by supplementation of easily digestible food, fresh water, barely water and supportive treatment.

3-Renal Abscess or Emboli

Definition:- It's lodging of bacterial emboli in the renal tissue after any bacteremia or septicemia then formation of abscess.

Etiology:- The origin of the emboli may be sporadic cases such as :

1-Supurative lesions in the uterus, udder, or peritoneal cavity.

2- Endocarditis.

Clinical Symptoms:-

1-Signs of toxemia and the primary causative disease are usually present.

2-Enlargement of the kidney may be palpable by rectal examination.

3-Spread of supurative emboli to the renal pelvis may cause a syndrome

of very similar to that of pyelonephritis.

Diagnosis:-

1-History of metritis or mastitis……etc. 2-Clinical symptoms

3-Laboratory diagnosis:- -Proteinuria.

-Blood and pus cells in microscopical examination of the urine.

-Culturing of urine may reveal presence of bacteria.

Treatment :-

1-Massive dose and long period of using of antibiotics 7-10 days according to the sensitivity test.

2-Correction of the primary diseases. 3-Supportive treatment.

4-Cystitis&Urolithiasis

Definition:- Cystitis means that inflammation of the urinary bladder which characterized by sings of abdominal pain and frequent urination.

Etiology:-1-Enterance of the infection during calving, dystocia or collection of the urine sample or using of urethral catheterization.

2-Extension of the infection from other parts of the urinary tract as in case of urethritis or nephritis.

3-Urine retention acts as a predisposing factor of cystitis.

Clinical Symptoms:-

1-Straining during urination which may be associated with grunting

2-Frequent urination

3-Signs of abdominal pain as arched back

4-The animal take the posture of urination for few moments after the end of the act of urination (from the pain)

5-The urine may tinged with blood or contains pus.

6-By the rectal palpation the wall of the urinary bladder is very thick with painful palpation.

Diagnosis:- 1-Case history and clinical signs

2-Rectal examination and urine analysis (presence of epithelial and pus cells).

Treatment :- 1-Analgesic and antispasmodic to relief the pain (Analgin-Buscopan-Glucolynamine…….etc.).

2-Irrigation of the urinary bladder by saline solution with antibiotics and administration of systemic antibiotics.

NB: Urolithiasis as mentioned before in part (I) Small Ruminant Medicine

5-Acute Tubular Necrosis ATN

[Tubular Nephrosis]

Definition:- It's non-inflammatory degeneration and necrosis of the renal tubules.

Etiology:-

1-Renal ischemia - salmonellosis 2-Ketosis

3-Sever septic process 4- Hypophosphatemia

5- Nephrotoxins which may occur with nephrotoxic agents as bile or hemoglobin.

6-Heavy metal nephrotoxic agents e.g. ; arsenic, mercury, lead & cadmium.

7- Some antibiotic if it taken for long period as. : Tetracyclines - Gentamycin, sulphonamides and Aminoglycosoides specially if it taken in dehydrated cattle.

8- Some plant poisoning.

Clinical Symptoms :- Together with the sings of the main cause :-

1-Marked depression, recumbence, anorexia and polyurea.

2-Haematouria and dehydration.

Diagnosis:- **1-**History and clinical symptoms.

2-Laboratory diagnosis.

A)-Blood analysis:

1-Elevated BUN and creatinine

2-Hypocalcemia, hyponateremia and hypokalemia

B-Urine analysis: 1-Low specific gravity of the urine.

2-Proteinuria. 3-Large number epithelial cells in the urine

Treatment:- Dealing wit the main cause according to the etiology

1-Mainly directed toward correction of the epithelial regeneration which manifested by (increase the specific gravity – reduction of serum BU.N. and creatinine).

2-Removal the animal from the source of toxins.

3-Restoring blood volume with fluid therapy containing isotonic amounts of calcium, chloride and sodium.

4-Fuosemid (Lasix) as diuretic 1mg/kg I/V. until urine flow is observed.

Chapter No.(5)

Nutritional And Metabolic Disorders

1-Rickets 2-Osteomalecia

3-Copper Deficiency

4-Hypophosphatemia

5-hypocalcemia

6-Hypomagnesemia

7-Ketosis

8-Downer Cow Syndrome

8-Fat Cow Syndrome

10-Cobalt,Iron,Vit.E &Selenium, Zinc and Vit.D &A Deficiency

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1-Rickets

Definition:- It is a nutritional deficiency disease of the young growing calves, characterized by defective calcification of the growing bones.

Etiology:- Deficiency of one or more of calcium, phosphorous and vitamin-D

A)-Calcium deficiency:-

1-Primary calcium deficiency due to lack of calcium in diet (rare).

2-Secondary calcium deficiency due to marginal calcium intake with high phosphorous intake.

B)-Phosphorous deficiency:-

1-Phosphorous deficiency usually primary under field condition but it can be exacerbated by deficiency of vitamin-D or excess of calcium.

C)-Vitamin-D deficiency:- **(See part (I) Small Ruminant Medicine)**

1-Usually due to lack of ultraviolet solar radiation of skin caused with deficiency of performed vitamin-D complex in diet.

2-Vit-D2 (calciferol) present in sun cured hay.

3-Vit-D3 (Cholecalciferol) produced by natural radiation with the ultraviolet. 4-Vit-D4, D5 in the fish oil.

Pathogenesis:- 1)-Deficiency of calcium, phosphorus and vitamin -D result in defective mineralization (calcification) of bone.

2)-See Vitamin D deficiency in part (I) Small Ruminant Medicine

Clinical Symptoms:-

1-Stiffness in gait, lameness and tendency to be down.

2-Abnormal curvature of the long bones which usually forward and outward with arched back

3-Enlargment of the joints specially of the fore limbs.

4-Softness of jaw bones, which severely affected animals unable to close their mouth and tongue, protrudes with salivation and abnormal prehension and mastication.

6-Dental abnormalities (Loose teeth, easily broken teeth……etc.):

17-Dyspnoea if the bone of the ribs affected.

11-Hypersensetivity, tetany and recumbence in the final sever stage.

Diagnosis:-A-History and clinical symptoms.

B-Laboratory diagnosis.

1-Serum phosphorous level is below 3mg% (N = 5-7mg %)

2-Serum calcium level is below 8mg% (N = 10-12mg %)

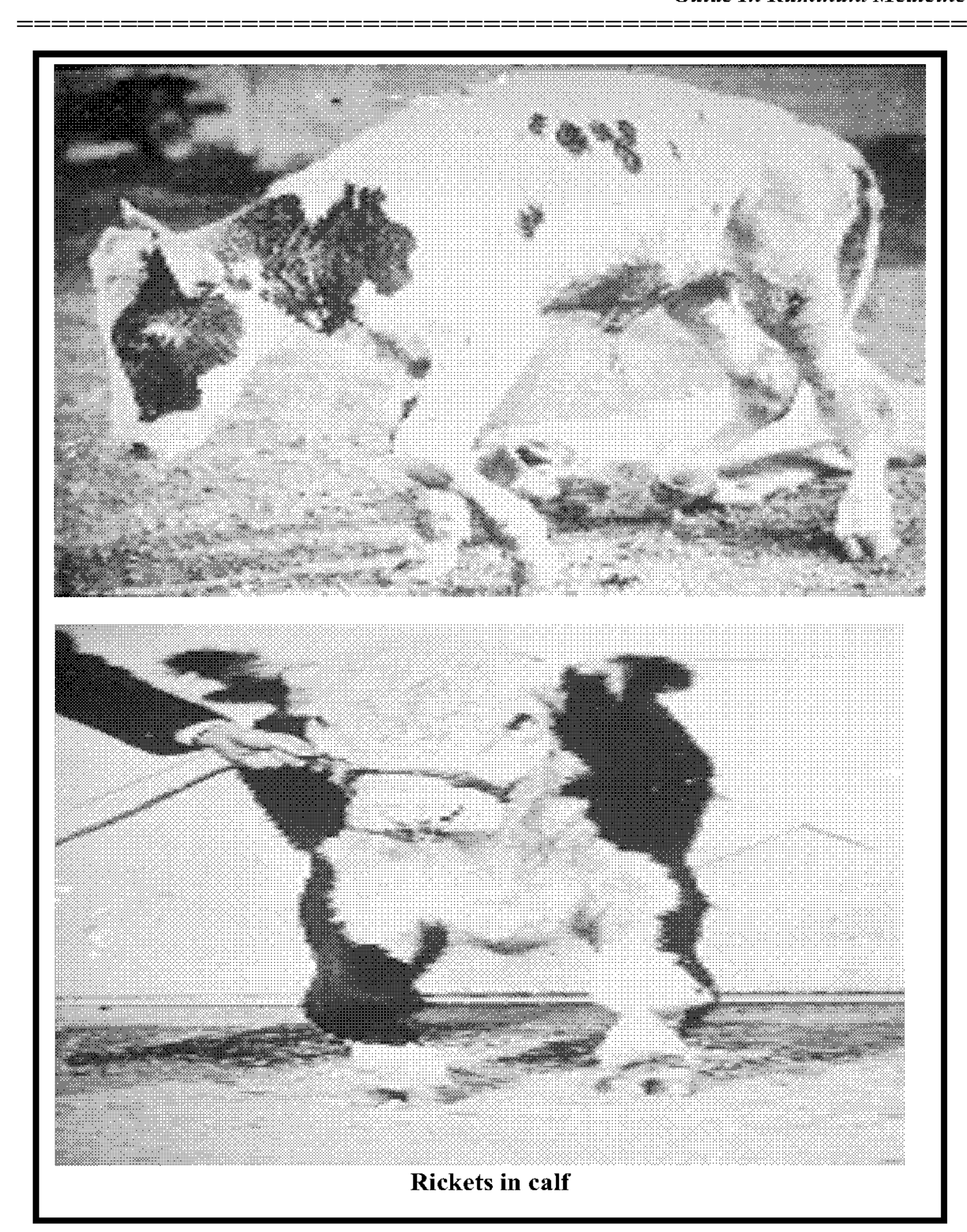
3-Serum 25-dihydroxy vit-D may be non detectable.

C)-Radiographic examination of bones and joints:

Treatment And Control :

1-Lesser deformities are recovered by the suitable treatment but the greater deformities usually persist.

2-Single parenteral I/M injection of 5000 IU /kg.Bwt- vit-D may meet the need of the animals for 3-6 months.



Rickets in calf

3-In the same time, animals must receive adequate calcium from lime stone, bone meal, molasses…etc. and phosphorous from bran, sodium acid phosphate, …….ect.

4-Exposure of animals to ultraviolet solar irradiation early morning and early evening.

5-Injection of calcium and phosphorous preparations.

6-Administerationof a dose of vitamin D in the late stage of pregnancy to the dame to avoid the occurrence of rickets in the newly born claves.

2- Osteomalacia

Milk lameness or Milk leg disease.

Definition:- Osteomalicia is a nutritional deficiency disease of the mature animals affecting bones and characterized by osteoporosis which manifested clinically by lameness and high susceptibility for bones fractures.

Etiology:- The same as for rickets except that the predisposing cause is not the increased requirement for growth as in case of young animals, but the drain of pregnancy and lactation.

Clinical Symptoms:-

A)-In the early stages :

The signs are those of phosphorous deficiency including :

1-Licking and chewing of inanimate objects

2-Infertility and lower of the reproduction rate

B)-In the later stages :

The signs are specific to osteomalacia including:

1-Painful condition of the joints and bones manifested in the animal by moderate lameness which always shifting from leg to leg [Milk lameness commonly occurs in heavily milking cows].

2-Arched back with crackling sound while walking.

3-Hind legs are most severely than the fore legs.

4-The bone easily fractured.

5-Abnormal mastication if the bones of the face are affected with dyspnoea if the bones of the ribs are affected

6-Dystocia when the pelvis bones are affected.

7-Final weakness leads to permanent recumbence and death from starvation.

Diagnosis:-

1-History and clinical symptoms.

2-Laboratory examination. Similar as in rickets.

3-Radiographic examination

Treatment :-

As mentioned before in rickets.

3-Hypomagenesemic Tetany

Whole Milk Tetany of Calves

Definition:- It's a nutritional deficiency disease with close similarity of lactation tetany and characterized clinical by hyperesthesia, tetany, and convulsion and characterized biochemical by hypomagnesaemia.

Etiology:- 1-The basic metabolic changes in calf tetany is hypomagnesaemia which accompanied in many cases by hypocalcaemia.

2-***Primary causes***:- The cause of hypomagnesaemia is the dietary deficiency of magnesium which exacerbated by high intake of calcium. i.e. 3-Milk in spite of it's low magnesium contents is inadequate source of the element (magnesium)for very young calves, so when the calves depend upon the whole milk only in the feeding system, it will be highly susceptible to hypomagnesmic tetany (so the name of the diseases is also whole milk tetany).

N.B. The hypomagnesaemic tetany in calves usually complicated with other many diseases under field conditions as enzootic muscular dystrophy (Vitamin E and selenium deficiency).

Incidence, occurrence and predisposing factors :

1-Most incidence in calves between 2-4 months of age.

2-In calves fed solely on diet of whole milk (Whole milk tetany).

3-More common in calves scour.

Clinical Symptoms:-

1-The calves apprehensive with constant movement of the ears.

2-Retraction of eyelids with nystagmus (rotation of eye ball).

3-Champing of jaw with grinding on the teeth.

4-Shaking of the head.

5-Hyperasthesia (hypersensitivity against external stimuli as movement, touch or light).

6-Fine muscular tremors with involuntary passage of urine and feces.

7-Tetany and convulsions after falling down [in the form of clonic convulsions] with accelerated pulse.

8-Temperature → within normal range but may be elevated due to muscular convulsions.

Diagnosis:-

A)-History and clinical symptoms

B)-Laboratory diagnosis:

1-Low serum magnesium level below 0.8 mg% [N=2-2.5mg/dl.].

2-Detection of magnesium level in the other body fluids as CSF (very low).

3-Detection of calcium: magnesium ratio, we found it is more than 80: 1 while normal ratio 70: 1.

4-Detection of urine magnesium level (very low).

Treatment:-

1)-Similar to that of hypocalcaemia due to the hypomagnesaemia usually associated with hypocalcaemia (See hypocalcaemia and hypomagnesaemia in this part of Large Ruminant Medicine).

2-Food supplementation with magnesium oxide or magnesium carbonate.

** 1gm daily magnesium oxide for calves up to 5 weeks of age.*

** 2gm daily magnesium oxide for calves up to 5-10 weeks of age.*

** 3gm daily magnesium oxide for calves up to 10-15 weeks of age.*

3-Tranquilization or using of ataractic drugs as chlorpromazine HcL to avoid death from respiratory paralysis.

Prevention :-

Supplementation of magnesium source in the diet especially in the first 10 days of age and continue until 15 weeks of age as mentioned in the treatment.

4-Copper Deficiency

Falling disease

Etiology, pathogenesis and diagnosis similar to that of sheep and goat (see part (I) Small Ruminant Medicine) in addition to:-

Clinical Symptoms:-

A)-General syndrome:

1-Unthriftness, loss of milk production and anemia and the coat color is affected.

2-Change I the color of the coat specially in the black or gray animals

3- Poor growth, increased tendency for bone fracture and sometimes chronic diarrhea in calves.

4- In some cases of calves ataxia develops after exercise due to demylenation of C.NS.

B)-Specific diseases:

(I)-Falling disease of cattle:

*Cow in apparently good health condition throw up their head, bellow and fall, sudden death after struggle and falling on their sides.

(II)-Unthriftness (pine) of calves.

*Progressive unthriftness, emaciation and grayness of hair especially around the eyes in black cattle (spectacled appearance).

(III)-Peat scours (teart) of cattle:

1-Persistent diarrhea with passage of watery, yellow green to black feces with an offensive odor.

2-The feces are released without effort, often without lifting the tail.

3-Sever emaciation although the appetite remain good.

4-The hair coat is rough and depigmented manifested by reddening or gray spots, especially around the eyes in black colored animals.

Diagnosis :-

A)-Case history and clinical symptoms

B)-Laboratory diagnosis:

1-Estimation of plasma and liver copper.

-Plasma copper level below 20µg/dl [N = 20-60 µg/dl].

-Liver copper value below 30mg/kg dry matter in cattle [N = 100 or above 100mg/kg. DM].

2-Estimation of copper contents of hair: N = 6.6 – 10 mg/kg.

3-Estimation of copper containing enzymes:

-Estimation of erythrocyte superoxide dismutase value range from 21-6 IU/mg hemoglobin in hypocuprosis.

-Estimation of copper – protein complex [ceruloplasmine].

*Ceruloplasmin contains more than 95% of the copper which circulating in the blood. Normal plasma ceruloplasmine level is 120-200 mg/dl.

Treatment:-

1-Oral dosing with 4gm copper sulfate for calves 2-6 months of age and 8-10 gm for mature cattle. Given weekly for 3-5 weeks.

2-Single parenteral injection of copper glyconat 400 mg for cattle by I.M or s/c.

3-Supplementation of the diet of the affected animals with copper (copper portion is about 3-5% of the mineral mixture).

Prevention:-

1-Supplementation of the diet with the minimum requirement of copper 10mg copper /kg matter.

2-Oral dosing or dietary supplementation of copper sulfate 4gm/cattle.

3-Mineral mixture of salt licks containing 2% copper sulfate for cattle.

5-Copper oxide needles:-

A-Fragments of oxidized copper wire up 8 mm in length and 1/2 mm. diameter for oral dosing considered as one of the most effective and safest methods for control of copper deficiency in ruminants.

B- The needles retained in fore stomach and abomasum for 100 days or more and copper slowly released, absorbed and stored in the liver.

5-Hypophosphatemia

Synonyms:-

1- Post-parturient haemoglobinuria. 2- Phosphorus deficiency.

Definition:- It's a metabolic disease characterized by pica, poor growth, infertility and in late stage with osteodystrophy and haemoglobinuria (due to increasing of fragility of RBCS).

Etiology:-

1- Primary under field conditions due to feeding on phosphorous deficient diet as barseem.

2-Vitamin – D deficiency or excesses of calcium.

3- Experimentally large dose of vitamin – A decrease the absorption of the phosphorus.

4-Enteritis or sever diarrhea which leads to decrease the absorption of phosphorus.

5-Impaction may be associated with hypophosphatemia due to malabsorption.

Epidemiology:-

1-In contrast to calcium deficiency, phosphorus deficiency is widespread under natural conditions.

2- Soil that contain excess of calcium, aluminum or iron that reduce the availability of phosphorus to plants, so the animals which fed on the plants

which cultivated in such type of this soil will suffering from phosphorous deficiency.

3- A primary dietary deficiency of phosphorus in dairy cattle within the first several weeks of lactation can result in the disease.

4- Under range conditions milking cows are most commonly affected.

5- The incidence is most common in animals at pasture during the barseem season.

6-High incidence in 3^{rd}. to 8^{th} lactation of high producing milk animals.

7-More common in barseem season [April to May].

8-In drought seasons but can also be a serious problem in housed cattle fed on the hay only.

9-Cattle and buffalo fed on a phosphorus deficient diet did not develop a detectable signs of phosphorus deficiency until they had been on a severely deficient diet for 6 months.

10-Secondary phosphorus deficiency due to excessive calcium intake or vit – D deficiency is of minor importance compared with the primary condition.

11- The diseases is more common in Barseem season because it is highly deficient in phosphorous.

12-Copper and selenium deficiency are predispose to haemoglobnuria because they are important as they protect the RBC, from orally acquired hemolytic agents [as saponine which may be presented in leaves of Alfa alfa or barseem].

Pathogenesis:-

1- Phosphorus is very important for development of bone and teeth, so its deficiency leads to abnormal development of the bone and teeth.

2- Phosphorus deficiency leads to deficiency in the 2-3 diphospho glyconate in the erythrocytes that leading to increase the fragility of the RBCS causing intravenous hemolysis and hemoglobinuria.

3-Inorganic phosphorus also play an important role in the metabolism of carbohydrates metabolism during muscular contraction, so phosphorus deficiency may leads to:-

A)-Recumbence of the cow or buffalo.

B)-Ruminal atony with bloat (free gas temany)

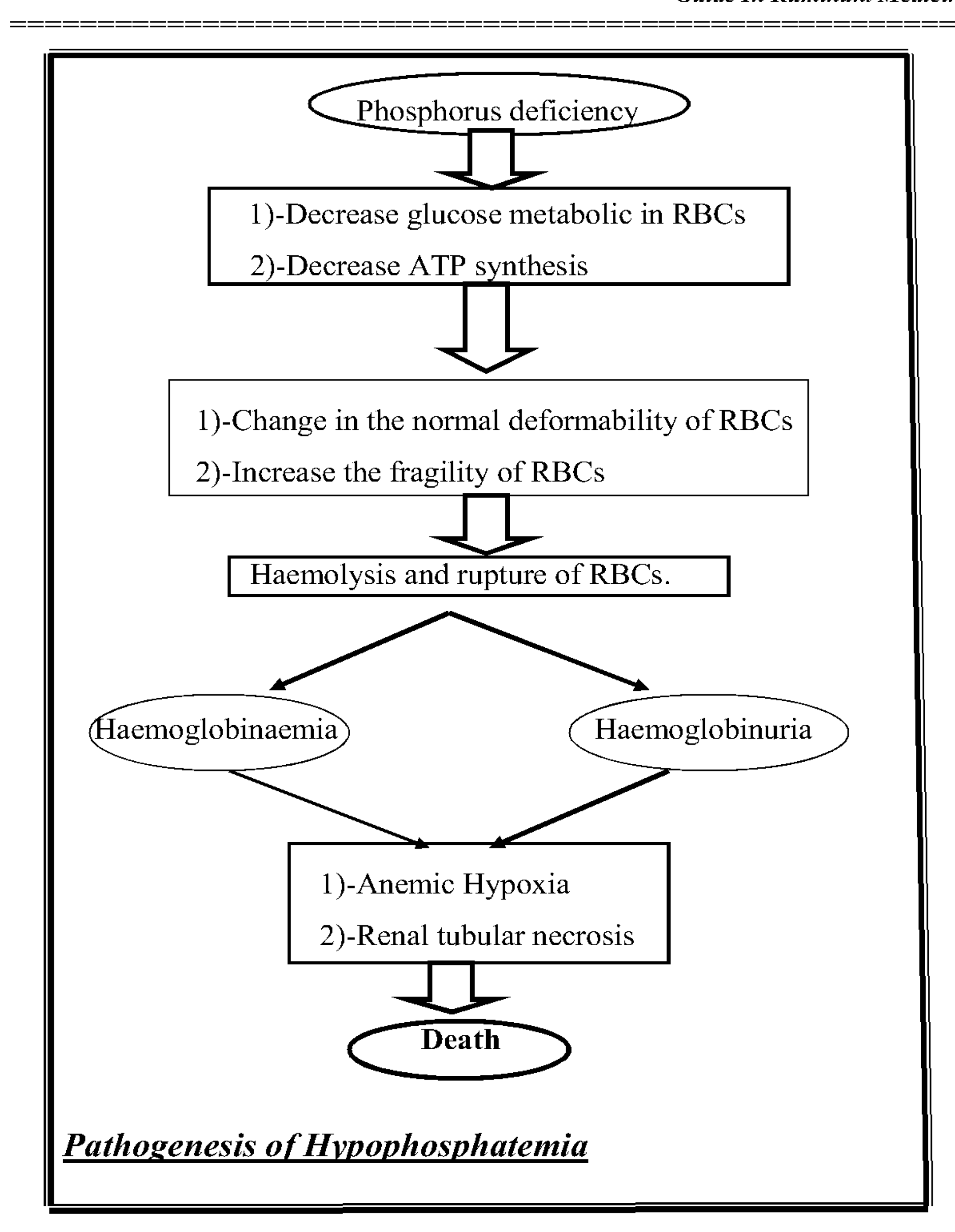
C)-General weakness

D)- Constipation (scanty feces with inappetance or anorexia).

4-Phophorus is very important for formation of the phospholipids portion in the milk, so phosphorus deficiency may lead to reduction in the milk production.

5-High milk producing cow or buffalo are more susceptible due to the lactation causes further depletion of the phosphorus in the milk.

6-Phosphorus is very important in vital processes in the RBCs as glucose metabolism in RBCs and ATP synthesis, so phosphorus deficiency leads to decrease in the metabolic processes that occurred in the RBCs. With decrease in the ATP synthesis with increase the RBCs fragility and hemolyesis resulting in haemoglobenuria and haemoglobenuria.



Pathogenesis of Hypophosphatemia

Clinical Symptoms:-

1- It leads to rickets in young animals and osteomalicia in adult.

2- Inappetence, loss of body weight, decreased milk yield and reduce the fertility. [Calving percentage drops from 70% to 20%].

3- Malocclusion of the teeth which may be resulted from poor mineralization of the teeth and weakness of the mandible.

4- More common in dairy cows under natural conditions while experimentally production of phosphorus deficiency in beef cattle indicate that several months on a deficient diet are necessary before clinical signs develop.

5- Abnormal stance, locomotion and recumbence with aletriophagia [pica].

6- Rough coat and the animal is anemic with poor physically conditioned.

7-Hemoglobinuria, dehydration, and the mucous membranes are pale in color.

8- Dry and firm feces and the body temperature may be rise.

9- Dyspnoea and tachycardia are common.

10- Jaundice may be developed in late stage [due to hemolytic anemia which may lead to hemolytic jaundice].

11- Sloughing of the tip of the tail or the digits has been observed occasionally or gangrene of such organs may occur.

12- Death occurs within few days usually due to anemic hypoxia or renal failure which resulted from renal tubular nephrosis.

13- Non fatal cases, the convalescence requires about 3 weeks and recovering animals often show pica.

14- Ketosis commonly occurs coincidentally with hypophosphatemia.

15- The urine is dark red- brown to black and turbid [in sever long standing cases].

16-Inappetance may appear in the form of tendency to eat the dry food than green food.

Diagnosis:-

(I)- Case history and clinical finding.

(II)- Laboratory diagnosis [clinical pathology].

1-low level of serum in organic phosphorus [N= 5mg%]..

2-Normal level of serum calcium level [N = 10mg%].

3-Low levels of copper in the serum and liver tissue of affected animals.

4- Numbers of RBCs and Hemoglobin are greatly reduced. [RBCS N= 5-8 millions/cum - Hb% N = 10-12mg%].

5-Turbid dark red–brown to black colored urine but not contains RBCS.

6-Heinz bodies may be present in the erythrocyte, due to saponine substance in barseem which leads to denaturation of the protein substances of the RBCs.

7- Billirubine high than normal.

3-Differential Diagnosis:-

*With diseases characterized by hemolytic anemia with or without hemoglobinuria as.

1- Leptospirosis

2- Chronic copper poisoning.

3- Cold water hemolytic anemia in calves.

4- Pyelonephritis due to coryn. renal.

5- Babesiosis.

6- Anaplasmosis.

7- Cystitis.

8-Water intoxication

NB.

*We can differentiate between haemoglobinuria and haematouria by centrifugation of the urine sample in a test tube , then if there is sedimentation (consists of RBCs) ,so it is considered as haematouria, while if there is no sedimentation and whole the column of the tube take the same red color(i.e. haemolysis of RBCs),so it is indication to haemoglobenuria.

Treatment:-

1- Transfusion of large quantities of blood is the only treatment required for saving the severely affected animal. 5 liters of blood / animal 450 kg bwt.

2- Fluid therapy to avoid the hemoglobinuric nephrosis.

3- Administration of sodium acid phosphate.

* 60gm in 300 cm. distilled water intravenously with the same dose s/c. (i.e 20%. Of sodium acid phosphate).

* Repeate the s/c. dose at 12 hours intervals for 3 times.

* Similar dose orally.

* Bone meal 120 gm orally (on the ration) twice daily or other source of calcium and phosphorus for 5 days. [Expensive treatment].

* Vit. D injection is recommended to enhance absorption of phosphorus.

* Hematonic drugs is recommended as R/ Adenoplex fort injection.

* Catosal I. V or IM up to 100 cm daily.

* Tonophosphan. I.v or I. M up to 20 cm. daily or Phospho 20.

4- Ketosis is one of common complication due to defect in glucose metabolism in the RBCS, so drugs needed for treatment of ketosis may be included. (R/ Dextrose 5% I.V).

Control:-

(I)-Providing of the ideal requirement of phosphorus as 15 up to 40 gm / day / animal. [Generally 22 gm / animal which produce about 7.5 kg milk /day].

(II)-Providing of bone meal or mineral mixture [up to 1kg / lactating cow /week].

(III)-Providing of copper in the ration to avoid it's deficiency.

6-Parturient Paresis [Milk Fever]

Parturient Hypocalcaemia

Definition:- *Parturient paresis is a metabolic disease occur most commonly in cows 48 hours after parturition, but it may occurs several weeks before or after parturition characterized clinically by :

-Generalized paresis. - Depression of consciousness.

-Muscular weakness and recumbence

-Characterized biochemical by: Hypocalcaemia.

Etiology:- -The main cause of parturient paresis is depression of the level of ionized calcium in the tissue fluid which usually attributed to imbalance between out put of calcium in the colostrums and the calcium in flux [input] to the extracellular pool from the intestine [by absorption] or from the bone (by mobilization or resorpition)

A- Factors leading to increase the calcium output:-

1- Producing of large amount of colostrums AS: cow that produce 10 kg milk colostrums [each kg. contains 2-3gm of calcium]. So the animal loss about 23 gm. Of calcium per each milking, this about nine times as much calcium as that present in the entire plasma calcium pool in cow (8-12mg/dl.).

2- Defects in the absorption of calcium through intestine due to sever diarrhea that leading to loss of large amount of calcium with the feces.

3- Excessive secretion of calcium in the urine due diuresis.

4- Increased calcium bone mineralization of the fetus.

B- Factors leading to decrease the calcium inflow:-

1- Impairment of absorption of calcium from the gut due to the fetus press on the gut especially in the late stage of pregnancy.

2- Insufficient mobilization of calcium from bone due to aging or senility.

3- Feeding on ration deficient in calcium.

N.B. * Some theory thing that dysfunction of parathyroid gland may lead to hypocalcaemia that attributed to dysfunction of parathyroid hormone which responsible for regulation of calcium level in the blood.

*Vit. D deficiency may lead to hypocalcaemia due to the important role of vit. D in absorption of calcium.

* Increasing of calcitenon or thyrocalcitenone hormones may lead to hypocalcaemia. As these hormones considered as calcium depressing factors.

→Estrogen hormone decreases the ionization of calcium→ Hypocalcaemia.

Occurrence And Predisposing Factors:-

1- The disease is most commonly occur in high producing adult lactating cattle [3-8th calving] i.e. 5-10 years of age [aged or senile animal].

2- Jerseys cows are most susceptible about 33%.

3- Hereditary causes may be involved [6-12%].

4- Complete milking in the first 48 hours after parturition or sucking normally by the calf may consider as precipitating factors because the colostrums contain very high amount of calcium.

5- ***Parturient paresis may occur in 3 stages of parturition cycle***:

A)- It may occurs in the last few days of pregnancy or at the act of parturition

but rare cases occurs several weeks prepartum.

B)- It may occurs in the last few hours of pregnancy or at the act of parturition and it's usually associated with uterine inertia due to the important role of calcium ion in maintenance of muscle tone of the uterus.

C)-The most common cases occurs the first 48 hours after parturition and the danger period may extend up to the 10^{th} day postpartum.

6- Occasional cases may occur 6-8 weeks after commencement of lactation

7- Heifer may be affected [rare].

8- Parturient paresis may occurs in the animal which suffering form:-

A)-Long period of starvation 48 hours with decrease the calcium level in the ration. B)-Undue fatigue and excitement C)-Persistent untreated diarrhea.

9- High incidence in winter and during feeding on bad quality roughage.

10- High protein in diet or feeding on diet containing oxalate which impairs the absorption of calcium leading to hypocalcaemia.

11- Buffaloes less susceptible than cows.

12- Sub–clinical hypocalcaemia may occurs in the first few weeks of lactating period and it's usually associated with ruminal stasis and anorexia leading to disturbance in energy balance of the body.

13- Injection of certain drugs specially aminoglycoside as gentamycine dihydrostreptomycin, and neomycin which reduces the ionization of the calcium may enhance the occurrence of hypocalcaemia.

14- Mortality: up to 3.5 - 8.5%

15- High calcium level in the diet (100 gm ca+ / day of cow 500 kgBwt.

especially in the dry period) will increase the incidence of milk fever ,as these leads to inhibition of the parathyroid gland to produce parathyroid hormone which is very essential for regulation of the serum calcium level[in prepartum or dry period] ,so the animal can not adapt to calcium loss postpartum (N= calcium requirement in dry period up to 30 gm/day to meet daily maintenance and fetal demands of calcium in the late gestation i.e. 6gm calcium /100 kg Bwt.).

16- High phosphorus in diet increase the onset of milk fever as it increase the concentration of phosphorus in the blood which is inhibitory to the renal enzyme which catalyze the production of 1,25 dihydroxycholelalcipherol (Vitamin D) which when it is decreased it leads to reduction of the intestinal calcium absorption mechanism prepartum.

17- Aged animals or senile animals are highly susceptible that due to:

A)-The ability of adaptation against calcium loss in aged animal is less than that of the heifer.

B)-Numbers of osteoblast cells are decreased with the age [these cells are very important for mobilization of calcium from bone to the blood].

C)-The tissues and intestinal receptors of vitamin – D are reduced with age, so the absorption of calcium is reduced with the age leading to increasing the incidence of disease.

D)-Decrease the level of hydroxyprolen (with the old age) which is very essential for mobilization of the calcium from the bone to the blood.

Pathogenesis:-

*Increase calcium outflow

*Decrease calcium inflow

Parathyroid dysfunction

Decrease the blood calcium level

Insufficient PTH for controlling the calcium level in the blood

Hypocalcaemia

-Decrease secretion of acetyl cholin
-Decrease the muscular tone

Ruminal stasis

Muscular weakness

Decrease the blood flow to the skeletal muscle and alimentary tract

Downer cow syndrome

Pathogenesis of Hypocalcaemia

Clinical Symptoms:- "3 stages"

A- Prodromal or Excitement stage.

B-Semi–comatoized stage or stage of sternal recumbence.

C- Comatose stage or lateral recumbence stage.

A- Prodromal or Excitement stage.

1- Excitement and tetany with hypersensitivity.

2- Muscular tremors of the head, neck and limbs.

3- Protrusion of the tongue from the mouth and grinding on the teeth.

4- Disinclination to eat or to move.

5- The animal is ataxic, fall on the ground easily and in going down the hind legs are extended stiffly.

6-Vigorous licking of the skin.

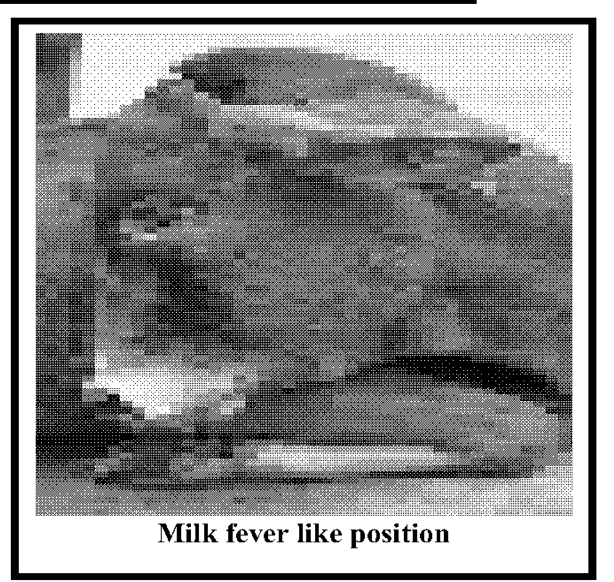
7- Normal temperature or may be slightly above normal due to excitement.

B- Semi–comatoized stage or stage of stenal recumbence:

1- Depression of consciousness and dry muzzle.

2- Cold skin with normal or subnormal temperature.

3- Sternal recumbence with characteristic position which is the head is turned laterally toward flank and resting on the shoulder.



Milk fever like position

4- Dilated eye pupil and absence of pupilary light reflex.

5- Ruminal stasis and constipation (scanty feces) or tempany [characteristic due to recumbancy]. 6- The animal appears drowsy and depressed.

7- The pulse is increased and weak (80/minute).

8- When approach to the cow it may open it's mouth with extend head and neck and protrude the tongue from the mouth as response to apprehensive or feared animal which is unable to raise.

9- Tetany of limbs disappeared and the animal unable to rise.

10- Venous pressure is low, so it's difficulty to prepare of jugular vein for intravenous administration. 11- Absence of anal reflex with dilated anus.

C- Comatoized stage or lateral recumbence stage:

1- Lateral recumbence with complete flaccidity of the muscles.

2- Absence of all reflexes. 3- Heart rate up to 4o/minute.

4- Bloat usually due to posture of recumbence.

5- It may impossible to raise the jugular vein.

6-Death within 12-24hours in untreated cases and the animal may die from cessation of respiration or during a convulsion.

N. B: A- If hypocalcaemia is associated with hypomagnesaemia we found:

* Trismus due to convulsion of muscles of mastication.

*Hypersensitivity is extended beyond the first stage.

* Increased in rate and intensity of respiration and heart rate.

B- If hypocalcaemia associated with hypophosphatemia we found response to calcium therapy except the animal can not raise.

C- If hypocalcaemia occurs in the few hours before parturition or at the act of parturition we found - Uterine prolepses -Dystocia. - Uterine inertia. But intravenous injection of calcium borogluconate will lead to normal parturition.

Diagnosis:- (A)-History and clinical symptoms.

(B)-Laboratory diagnosis [clinical pathology].

1- Depression of total serum calcium level [N= 8-12 mg%].

2- Glucose level may be lower than normal if hypocalcaemia associated with ketosis or it may higher than normal if the animal is aged.

3- Magnesium is low than normal especially if the animal present in pasture feeding system [N= 2.5 mg/dl].

4- Phosphorus is lower than normal [N= 5-7 mg /dl].

5- Neutrophillia. – Eosinopenia – Increase the PCV. and- Lymphopenia which indicates to hyperactivity of suprarenal gland so it's ensure that stress factors play a role in the pathogenesis of milk fever.

6- Increasing of some enzymes due to damage of the muscular cell membranes as Creatine Phasphokinase (CPK) or Glutamate–Oxalacotate Transaminase enzyme (GoT).

C- Diagnostic Therapy: - By response to intravenous injection of calcium borogluconate or other calcium preparations.

D- Differential Diagnosis

1- Hypomagnesaemia * Hypersensitivity with long period

*No response to calcium therapy. *Low magnesium, less than 1.2 mg/

dl. *Increase the intensity of heart sound.

2- Hypophosphatemia: - *Bloody urine

*Partial response to calcium preparation and the animal remain unable to rise.

*Low level of phosphorous in blood.

3- Aspiration pneumonia: * History of drenching the animal.

*High pulse rate and the animal lies on its side.

*Very clear respiratory sings.

*Regurgitation of food and water from the mouth and nostrils.

4- Coliform mastitis: *Changes in the udder and milk characters.

5- Ketosis: *Offensive or acetone odor of mouth.

*Positive (Rothar`s) sodium nitroprosside test

*No response to calcium therapy.

6- Downer cow syndrome: *Variable response to calcium, phosphorus and potassium therapy.

*Long course about 1-2 weeks. *The animal eats well.

7- Injuries in the hind quarter: *The animal eats and drinks normally

*The animal unable to raise but try to stand on the fore legs.

*No change in temperature, pulse or respiration.

Treatment:- (I)-Nursing or managemental treatment.

(II)-Chemotherapy.

(I)-Nursing or managemental treatment.

1- Feeding by stomach tube.

2- Turning of the animal or using of trochar and the canula to avoid tempany.

3- Avoid complete milking for 48 hours and the complete milking returned gradually.

4- Massaging of the legs and bony prominent to avoid leg dysfunctions.

(II)- Chemotherapy:

A- Treatment of parturient paresis by parenteral injection of the calcium preparations is the standard practices:

R/ Calcium borogluconate [drugs of choice] 100- 200 gm 20-30% solution i.e 400-800 ml 25% solution. I. V. or S/c.

The best rout is that intravenous or dividing the dose into 2 halves. One IV. and the other s/c.

(R/ Cal-D-Mag. or R/ Calbormag . R/ Calphomag.)

B- Signs of response to specific therapy:

1- Blenching with increasing of the intensity and rate of the pulse.

2- Tremors of the hindquarters and the tremors may extend to the body and head and neck.

3- Sweating of the muzzle and coat and the stool is characteristics is hard and covered with mucous and may contains flecks of blood.

4- Urination may follow rising of the cow.

5-The animal raise spontaneously or with the assist

(C)- We must follow or monitoring the heart rate during injection of the calcium especially intravenous, if the heart rate suddenly increased we must stop the injection for period then return again i.e. it's preferable to inject for 10 minutes not more and repeat it.

(D)- Injection of: - → Dextrose 20 % 500 ml I. V.

→ Sod. Acid phosphate 200 ml 15% sol. I.v

→ Magnesium sulphate 15 % solution 200-300 ml s/c

it's preferable to give preparation contains calcium, phosphorous and magnesium. As cal- D mag., or cal phomage….etc.

(E)- Injection of calcium preparation should be slowly to avoid the bad effect of calcium on the heart which may lead to heart block [bradycardia followed by tachycardia up to 180 Beats/min i.e. fatal heart beat]

*But Atropine preparations can be used for abolishing the resulting arrhythmia.

(F)- Avoiding injection of calcium if the temperature more than normal, so we must make to bring the temperature below 39C^{o} then injects the calcium preparations. (G)- Udder inflation may be used but its old fashion.

<u>Prevention And Control:-</u> 1- Avoiding any alimentary tract stasis or diarrhea to avoid any disturbances in absorption of calcium through the intestine specially in late stage of pregnancy.

2- Feeding on ration rich in phosphorus and low in calcium in the late stage of pregnancy [dry period] to stimulate the parathyroid gland and enhance the mobilization of calcium from the bone.

3- Feeding on ration rich in calcium in the postpartum period.

4- Milking before calving to avoid sudden fall of the serum calcium level after parturition.

5- Avoiding diet high in protein to avoid stimulation of maximum milk

production after parturition.

6- Oral administration of calcium in the form of calcium jell : in 3 doses.

1st dose → 24 hours before parturition.

2nd dose → 1-2 hours before parturition.

3rd dose → 10-14 hours after parturition.

7- Administration of vitamin -D2 early and vitamin -D3

- Orally: 20 millions I. U. daily for 5 days before parturition.

- Injection: 10 millions I. u. [250mg] and it can be repeated within interval 8 days before parturition if necessary i.e. if cow not calved in the expected time. (40 millions I. u. is the lethal dose which may lead to vitamin D toxicity.)

8 Avoiding of risk or stress factors.

9-Feding on "***ammonium chloride***" in grain 25 gm. And increased to100 gm. At calving gives good results that due to ammonium chloride gives acidic food which enhances mobilization of calcium form the bone and ionization of calcium which resulting in increasing of the total plasma calcium level.

NB-:-Contraindication to inject the calcium preparations intravenously rapidly because the calcium leads to:-

*Increase the varicosity of the blood

*Has vasoconstrictor action

*Has a direct effect on the heart

So, injection of the calcium preparations rapidly may lead to sudden death

7-Hypomagnesemia

Synonyms : 1- Lactation tetany. 2- Grass tetany.
3- Wheat pastures poisoning. 4- Grass staggers.

Definition:- It's highly fatal disease of all classes of the ruminants specially in lactating cow, characterized biochemical by hypomagnesaemia and hypocalcaemia [maybe] and clinically by hyperesthesia, clonic muscular spasm and convulsion and death which mainly due to respiratory failure.

Etiology:- 1-Feeding on diet deficient in magnesium may considered as the main cause because of the magnesium homeostasis or the ability of the body to adapt against magnesium deficiency is low, so the body depends mainly upon the magnesium intake in the diet. (Exogenous source).

2-High milk production may consider as predisposing factor of hypomagnesaemia as the milk contains large amount of magnesium [12 mg Mg/kg milk].

3-Feeding on diet intoxicated with nitrogen or urea or with potassium as fertilizer which impairs the absorption of magnesium in the fore stomachs.

4-Gastrointestinal disturbances which may interfere with the absorption of magnesium especially if it associated with feeding on young grass as wheat pasture which are rich in potassium and low in sodium.

5-Long period of starvation may sufficient to cause hypomagnesaemia specially if there is cold other.

6-Excitement or moving of the herd may lead to hypomagnesaemia that due to excitation which may lead to secretion of large amounts of epinephrine which play an important role in falling of serum magnesium level.

Incidence And Occurrence:-

1-Hypomagnesemia is more common in first 2-4 months of lactation specially in senile or aged and highly producing cows[4-7 years of age].

2-Morbidity up to 12% while mortality extremely high and the animal may found dead due to short course of the disease.

3-High acidic soil which may lead to increase the aluminum concentration that impairs the magnesium uptake by the plants resulting plant deficient in magnesium.

4-It occurs mainly in cows but sheep and goats also susceptible.

5-Magnesium requirements are:-

A)-1-1.3 gm Mg /kg. of dry matter for pregnant cow.

B)-1.8 – 2.2 gm Mg /kg. of dry matter for lactating cow.

6-Most clinical cases of the disease have serum magnesium level below 1mg% compared with the normal level in the cattle 1.7-3mg/dl.

Pathogenesis :-

1-Low magnesium intake less than the normal requirements of the animal leads to lowering of the serum magnesium level. Causing Hypomagnesaemia which leads to hyperesthesia [hypersensitivity to the external stimuli].

2-Hyperethesia occur due to: the magnesium is very essential in the

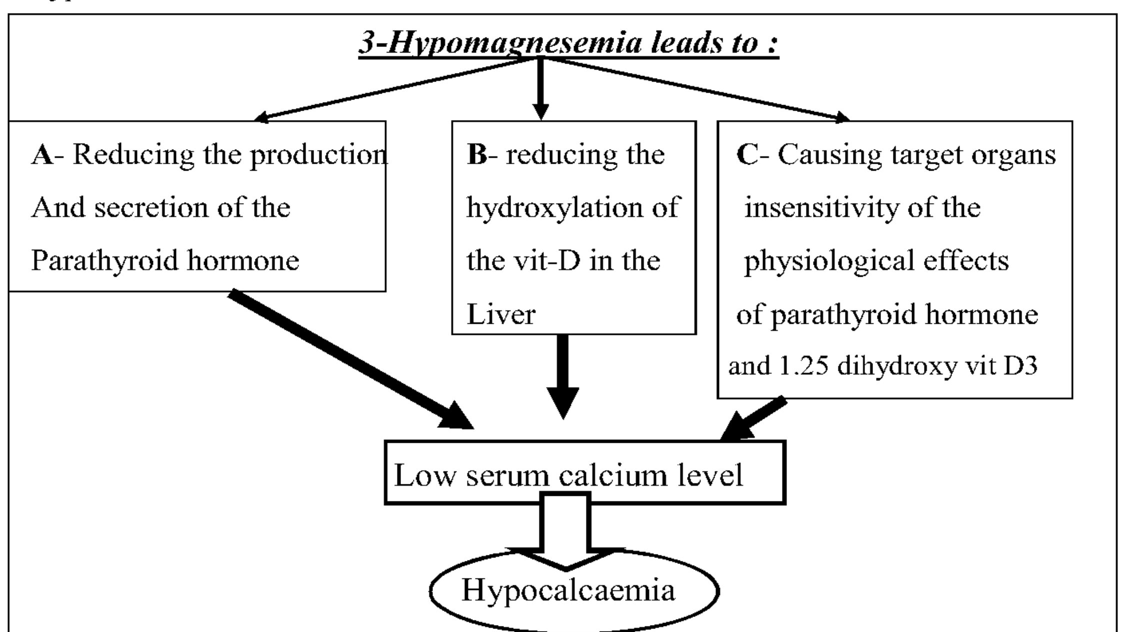
impulse transmission in the neuromuscular system as it affect on :

A-Releasing of acetyl choline which responsible for muscular contraction. [As calcium effect].

B-Activation of the cholinesterase which responsible for muscular relaxation.

C- Effect on the sensitivity of motor end plate and threshold of the muscle membrane.

So, Magnesium deficiency leads to disturbances of all mentioned process leading to tonic–clonic spasm and convulsion of the muscles with hyperesthesia.



So:-

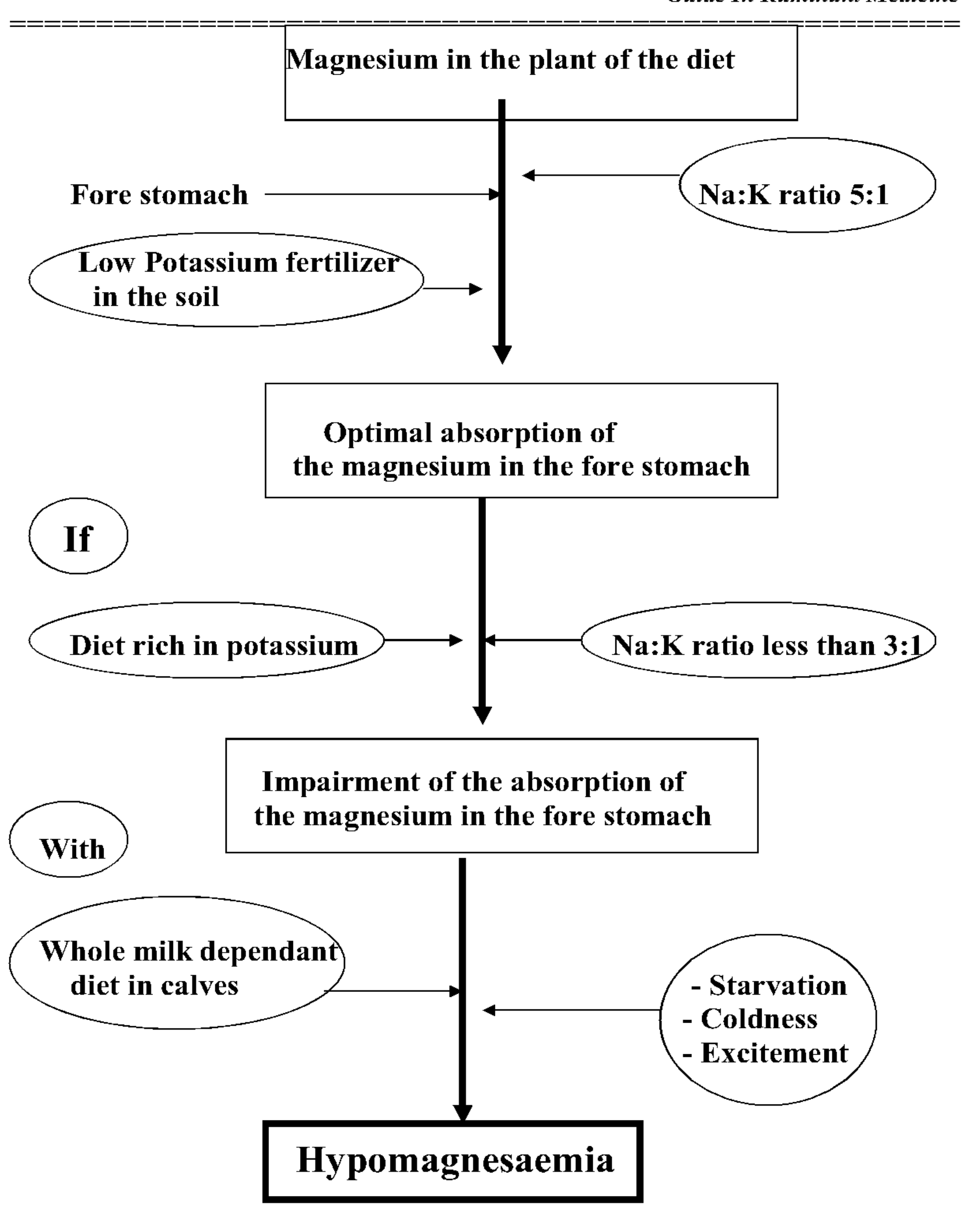
* Hypomagnesaemia usually associated with hypocalcaemia but not sever as that occurring in parturient paresis. So sub- acute or chronic hypomagnesaemia may acts as predisposing factors of parturient paresis.

4-In the adult ruminant, magnesium absorption occur in the fore stomachs and little absorption in the abomasums and small intestine and large intestine but the absorption through the large intestine not compensate the malabsorption in the for stomachs in case of hypomagnesaemia.

5-Magnesium is transported through the epithelium of the fore stomachs to the blood by an "active sodium – linked ATP-ASE dependent transport system"

5- Absorption of magnesium is influenced by Na : k ratio and the absorption is in optimal level when the Na : K ration is not less than 3: 1 ,So feeding on diet rich in potassium as feeding on the wheat pasture or plants from soil rich in fertilizers as potassium. Leads to impairment of the absorption of magnesium resulting in → hypomagnesaemia.

6- The following diagram shows the pathogenesis of the hypomagnesaemia.


Magnesium in the plant of the diet
Fore stomach
Na:K ratio 5:1
Low Potassium fertilizer in the soil
Optimal absorption of the magnesium in the fore stomach
If
Diet rich in potassium
Na:K ratio less than 3:1
Impairment of the absorption of the magnesium in the fore stomach
With
Whole milk dependant diet in calves
- Starvation
- Coldness
- Excitement
Hypomagnesaemia

Clinical Symptoms:- There four forms of the diseases

A- Acute lactation tetany. B- Sub – acute lactation tetany.

C- Chronic lactation tetany. D- Parturient paresis with hypomagnesaemia.

A- Acute Lactation tetany.-

1- The animal may be grazing at the time and suddenly cease to graze, and appear uncomfortable, and twitching of the muscles and ears is evident.

2- Hyperesthesia, Continuous bellowing and frenzied galloping.

3- The gait becomes staggering and the animal falls with obvious tetany of the limbs which is rapidly followed by clonic convulsions lasting for about minute.

4- During the convulsive episodes there is:

a- Opisthotenus, nystegmus and champing of the jaws.

b- Frothing in the mouth. c- Pricking of the ears and retraction of the eyelids.

5- Between episodes the animal lies quietly but sudden noise or touch may precipitate another attack of convulsion.

6- Body temperature is high after sever muscle convulsion (about 40-40.5C^{o}.)

7- Respiratory rates and pulse rates are also high.

8- The intensity of the heart sound is increased, so it can be heard some distance away from the cow.

9- Death usually occurs within 1/2 – 1 hour and the mortality rate is high because many die before treatment can be provided.

10- The response to treatment is good.

11- Death usually due to respiratory failure.

B- Sub – acute lactation tetany:-

1- The onset of the disease in this form is more gradually and the response of treatment is good and effective but there is a marked tendency to relapse.

2- Neglected cases may be advanced to recumbence and acute form.

3- Over a period of 3-4 days there are:

*Slight inappetence."The milk yield is diminished"

*Wildness expression of the face. * Exaggerated limb movements.

4- Muscle tremors and mild tetany of the hind legs and tail with an unsteady and abnormal gait may be a companied by retraction of the head and trismus (lock jaw).

5- Sudden movement, noise, application of restraint or insertion of needle may precipitate a violent convulsion again.

6- The animal with this form may be recovered spontaneously with a few days. 7- Spasmodic urination and frequent defecation are characteristic.

C- Chronic lactation tetany:-

1- There are many animals in the herd have low level of serum magnesium but not show clinical signs and there are sudden deaths.

2- Some animals may show vague syndromes including.

a- Unthriftness and dullness with low milk production.

b- Different appetite and may show one of the mentioned symptoms in the acute or sub acute form.

3- Chronic lactation tetany may also occur in animal which recover from the sub – acute form.

4- In lactating cow hypomagnesaemia may incremented to the occurrence of paresis and milk fever like syndrome that is poorly responsive to the calcium treatment.

D- Parturient paresis with hypomagnesaemia:-

1- There are paresis and circulatory collapse.

2- Flaccidity and dullness of parturient paresis replaced by hyperesthesia and tetany with slow response to the calcium therapy [see – parturient paresis].

Diagnosis:- (A)-Case history and clinical symptoms.

(B)-Laboratory diagnosis :

1-Estimation of the total calcium and magnesium levels in the serum and cerebrospinal fluid.

- Normal calcium. 8-12 mg% average 10 mg / dl.
- Normal magnesium 1.7 – 3 mg / dl average 2.5 mg / dl.

*And the symptoms of hypomagnesaemia not evident until the level of the serum magnesium fall to below 1.2 mg/dl.

*And sub- clinical hypomagnesaemia the magnesium level 1-2 mg / dl.

*And the average level at which the signs occur is about 0.5 mg / dl.

2-In wheat pastures poisoning of cattle there are:

- Hypomagnesaemia – Hypocalcaemia – Hyperkalemia.

* Normal C. S. F magnesium level in normal animal similar to that of plasma i.e. 2 mg % but in diseased animal collection of sample 12 hours after death the C. S. F. magnesium level is below 1.25 mg/ dl.

3-Because of the kidney is the major organ of homeostasis of the

magnesium so detection of magnesium level in the urine is good indicator. Specially for monitoring of the disease in a herd and also help in the early diagnosis of the diseases ***(Abd Elghany, H.A. 2000)***

4-Calcium level is below 5-8 mg/ dl.

5-Bone biopsy to estimate calcium: magnesium ratio 2: 1/2.

6-Therapeutic diagnosis : The animal response to calcium – magnesium preparation [cal-d- mag or calphomag].

(C)-Differential diagnosis :

1-Acute lead poisoning:-*Blindness * History of access to lead.

*Mania & no response to magnesium therapy

2-Bovine spongiform Encephalopathy (Cow made disease):

*Similar to sub-acute hypomagnesaemia but it's characterized by long clinical course.

3-Nervous form of ketosis *:* *Presence of characteristic ketonuria.

*Positive Rothar`s test *Response to glucose therapy

4- Rabies:- * Absence of tetany. *History of biting from rabid animal.

* Characterized by straining and ascending paralysis.

Treatment:-

1- The best example for treatment that using preparation containing magnesium and calcium salts in the lactation tetany and parturient paresis and that depend upon the result of biochemical tests and response to the treatment.

2- Magnesium salts solution is the drug of choice in lactation tetany.

3- S/C. injection of 200 ml 50% - solution of magnesium sulphate.

4- Preparation of calcium – magnesium solution. Can be given slowly intravenous 500 ml in cattle or buffaloes. R/ Cal-D-mag.

5- Intravenous injection of magnesium salts solution alone is very dangerous because it lead to cardiac embarrassment and medulary depression and leads to respiratory failure and death.

6- Magnesium gluconate at 15% solution can be injected 200- 400 ml I. V.

7- Oral administration of magnesium salts or feeding of magnesium rich – supplements is recommended after parenteral treatment.

8- In case of acute convulsions we can give ataractic drugs before the course of treatment (R/ Combelen – Rumbon – Neurazin – Neurel …etc.)

9- Rectal infusion of 30 gm of magnesium chloride in 100 ml solution is also recommended.

NB:-

* Because of the narcotic effect of magnesium ions it is contraindicated to use magnesium compounds alone because it may cause cardiac arrest ,so it's preferred to use calcium – magnesium preparation as (R/cal-dMag-or calphomage or calbormage……etc).

N B:-

*Administration of solution containing:-

10% magnesium sulphate. - 25% glucose. and 5% acid phosphate (sodium) give 200 ml s/c. and 100 ml I.V. give good result without relapse of the disease (*Indian vet. J. (1993) 70 (3) 247- 250).*

Control:-

1- Change the pasture during the grass tetany season.

2- Feeding of magnesium supplements: Feeding on magnesium preparation containing at least 87% magnesium oxide may prevent the seasonal falling in the serum magnesium level [60gm magnesium oxide / day].

3- The most effective amount is 120 gm of magnesium salts specially magnesium oxide per day but 180 gm. May causing diarrhea.

4- Magnesium salts are unpalatable, so it can be mixed with molasses in equal amount.

5- Magnesium phosphate 53 gm / Day is effective.

6- Magnesium salts – molasses mixtures diluted with water and spray on the hay is recommended.

7- Excessive amount of magnesium oxide 2-4% of the ration is toxic to calves causing diarrhea and the feces contains much mucus.

8- Magnesium bullets placed in the reticulum for slow liberation of constant traces of magnesium daily for long period as long as several months or even years.

8-Ketosis

Synonyms :-1- Acetonemia of cattle. 2- Post parturient dyspepsia.

Definition:-

Ketosis is a metabolic disease of the cattle characterized clinically by digestive and / or nervous signs and biochemical by: ketonemia, ketonuina, hypoglycemia and low level of hepatic glycogen.

Etiology:-

[mainly hypoglycemia]. And it may be :

1- Alimentary ketosis :

A – This occurs usually due to feeding on ketognic food as silage [highly ketogenic]. It contains high amount of the butyric acids (precursor of keton body or its ketogenic volatile fatty acid].

B- This type of ketosis is commonly sub clinical but it may predispose to the development of production or primary ketosis.

2- Production or primary ketosis.-

A- Usually occur in good or excessive body conditioned animals which have high lactation potential and are being feed good quality ration.

B- High protein in diet leads to greater butyric acid production in the rumen (ketogenic volatile fatty acid).

C- Excessive body condition and high protein in diet lead to excessive lipid mobilization and production of ketognic volatile fatty acids and ketogenic amino acids.

D-Feeding diet poor in carbohydrates.

E- Ketosis also may occur where excessive level body fat due to over fatness at calving time which inhibits dry matter intake, where there is acidosis due to the ration do not meet the energy requirements of high lactation potential cow that may predisposed to the disease.

3- Secondary ketosis:-

* Acetonemia may occur secondary to some conditions which associated with reduction in food intake at the post parturient period as abomasal displacement, metritis, mastitis or traumatic reticulitis. (This part is neglected in veterinary literature).

N. B.

-Adrenal dysfunction may lead to ketosis because of the hormones of the adrenal gland are very important in carbohydrate metabolism.

-Liver dysfunction → impairment of glucose production → hypoglycemia and ketosis.

4- Starvation ketosis :

A-This occurs in cattle that are in poor condition and that are fed poor quality feed stuffs.

B- Deficiency in propionate [glycogenic fatty acid] and protein from the diet and limited capacity of gluconeogenesis [production of glucose from non – carbohydrate source as amino and fatty acids] from body reserve.

5- Ketosis due to specific nutritional deficiency :

A-Specific dietary deficiency of cobalt leads to inadequate production of

oxalacetate from propionic acid, so it lads to defect in the oxidation of Acetyl – Co-A in tri–carboxylic acid cycle (kreb's cycle or TCA). So it leads to excessive production of acetoacetic acid and beta–hydroxybutyric acid (keton bodies). So cobalt deficiency is predisposed to ketosis.(See pathogenesis).

Incidence and occurrence:-

1-It's a disease of dairy cattle [especially that housed in winter and spring but rare in cows that are at the pasture] and it's prevalent in most countries where intensive farming system is practiced.

2-Incidence of sub-clinical ketosis is high especially in un-nourished herds.

3-Most cases occur in the immediate post – parturient period especially in the first 60 days of lactation, (about 90% of cases.)

4-Regardless the etiology it's more common in the 1st, month than the 2nd month of lactation period and occasionally occurs in the late stage of pregnancy.

5-It's occurred at any age beginning at first lactation within minimal level and reaches the peak specially at 4th lactation

6-It's economic importance is mainly due to loss of production which failure to return to full production after recovery.

7-It's more common in heavy producing animal and aged one 5-10 years of age.

Pathogenesis:-

1-Maintanance of glucose level in the blood is one of the main functions of the energy metabolic system in the body.

2-Ruminant absorb very little amount of the dietary carbohydrates. As hexose sugar because of carbohydrates are fermented in the rumen to fatty acids. Mainly acetate (70%), propionate (20%) and butyrate (10%) ***so***, Glucose need to gluconeogenesis (Creation of glucose from non carbohydrate sources) to maintain its normal blood level.

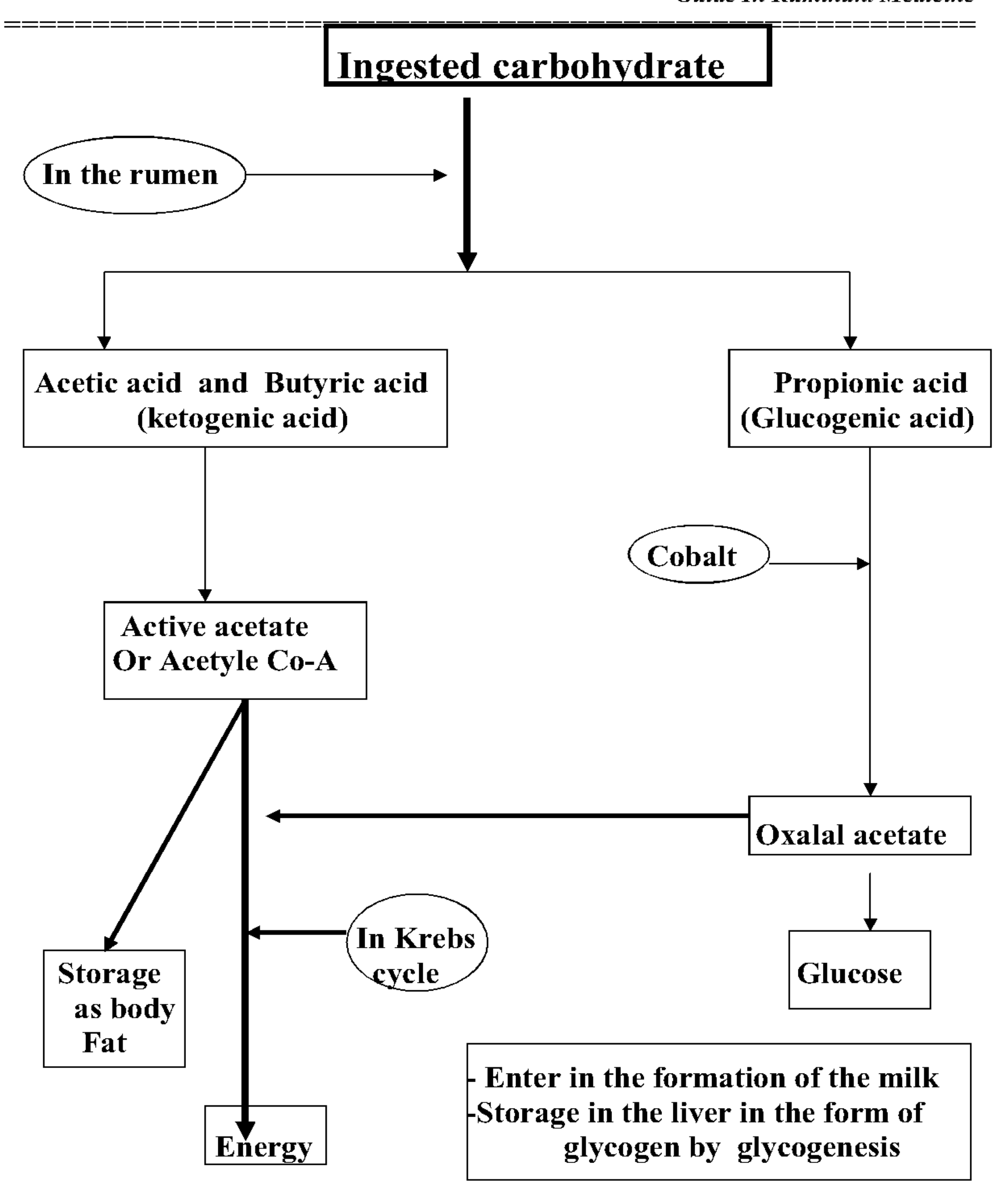
3-Glucogenic amino acids and propionate [glucognic fatty acid] are the major precursor of glucose in gluconeogenesis with glycol and lactate of lesser importance.

4-Propionate is produced in the rumen from starch, fiber and protein and it does enter the portal circulation and it's efficiently removed by the liver which is the primary glucose producing organ.

So: **:-** Increased availability of propionate in the diet can spare the hepatic utilization of other glucose precursor, and the production of propionate in the rumen. Is favored by high grain inclusion in the diet.

5-The most important keton bodies are → Acetoacetic acid - Acetone.
→ Beta–hydroxyl butyric acid

So:- Increase of such substance in the blood lead to ketonemia, and the following illustration show the normal pathway of the ingested carbohydrates and how and when the keton bodies are formed causing ketonemia and ketosis in the second diagram.



Normal Pathway Of Carbohydrate In Ruminant

Low carbohydrate in the diet

↓

Low production of propionate

↓

Low production of oxalacetate

↓ ↓

Impairment of Oxidation process Of acetyl Co-a To produce energy

Low level of glucose

↓

Hypoglycemia

↓

Increase fat catabolism

↓

Production of the keton bodies

↓

(1)Acetoacetic acid

(2)Beta hydroxybutyric acid

(3)Acetone

Pathogenesis Of Ketosis Clinical Symptoms:-

There are main 2 forms which are:-

A- Digestive or wasting or woody cow form.

B- Nervous form or typical form.

A- Digestive or wasting or woody cow form:-

1- It appears gradually and it's the most common form.

2- Gradual decreasing in the appetite and milk production over 2-4 days.

3- The animal appeared refuse to eat grains firstly then silage but may continue to eat the hay or may appeared depraved appetite.

4- Rapid loss of body weight and loss of subcutaneous fat and the skin loss its elasticity and the animal take. The "***woody appearance".***

5- Feces are firm and dry but serious constipation do not occurs.

6- There is moderately depressed and hangdog appearance and disinclined to eat and to move and may suggest the presence of mild abdominal pain.

7- Respiration, pulse, and temperature are normal.

8- Ruminal movement within normal numbers (3-5 / 2 minute) but the amplitude is low and with the progression of the disease the ruminal movement may be virtually disappear.

9- A characteristic odor of ketone [acetone odor] can be detectable in the breath and may in the milk.

10- The reduction in the milk yield may reach 25% but not return to the full normal again.

11- Some nervous manifestations may appear with the wasting form as slight staggering or partial blindness [rare].

B- Typical or nervous form:-

1-This form attributed to the production of isopropyl alcohol which produced as a result of excessive destruction of acetoacetic acid in the rumen.

2-Walking in circles. 3-Straddling in the gait or crossing of legs.

4- Head pushing or head pressing into the stanchion.

5- Apparent blindness. 6- Aimless movements and wandering.

7- Vigorous licking of the skin and inanimate objects.

8- Depraved appetite with chewing movements and salivation.

9- Hyperesthesia may be evident.

10- Nervous signs occur in episodes which lasting within 1-2 hours.

11- Affected cows may injury themselves during the nervous episodes.

12- Moderate tremors and tetany with in coordination of gait.

13- Coma and recumbence. 14- Ketonic breath odor is present.

Diagnosis:- (A)- Case history and clinical signs.

(B)- laboratory diagnosis:- from the clinical pathology we found:

1-Hypoglycemia, glucose level 20-40 mg% [N= 50-60 mg /dl].

2-Ketonemia, keton bodies in the blood 10-1000 mg/ dl [N=up to 10 mg %].

3-Ketonuria (keton bodies in the urine 80-1300mg/dl [N=up to 50-70 mg%].

4- Increase the level of plasma fatty acids due to acceleration of the processes of gluconeogenesis. 5- Milk ketones → 40 mg/ dl [N = 3-10 mg / dl].

6- Low level of hepatic glycogen.

7- High level of volatile fatty acids in the blood and rumen specially the butyric acid.

8-Low level of calcium up to 9 mg/ dl due to loss of base to neutralize the acidosis. 9-Increase the level of serum (SGOT).

10- High level of cortisone [but it may be increased under any stress conditions as fear, excitation, starvation……….etc.].

11-Change in leukocyte

(I)- Lymphocytosis → 50-80% [N= up to 60% ≃ 15-60%].

(II)-Eosinophilia → 15-40% [N= 0.6%].

(III)-Neutropenia → 10% [N = 33 -75%].

12-Detection of ketonuria by "Rother's" or "sodium nitrobrosside test".

(C)- P. M. lesions:- 1- Fatty liver infiltration. 2- Enlarged adrenal gland.

(D)-Differential diagnosis:-

1- Traumatic reticulitis: *Moderate fever.

*No relationship with recent calving. * Complete ruminal stasis.

* Detected by pain tests (positive) *Blood examination (Neutrophillia. And normal blood glucose level.)

2- Abomasal displacement: * No – response of treatment of ketosis.

*Occurs at calving. * Intermittent anorexia with pasty feces.

* Abomasal sound may be audible at lower left abdominal area.

* Normal glucose level.

3- Vagus indigestion: * History of improper diet or trauma.

* Usually lead to secondary ketosis. * Moderate recurrent bloat.

*Marked alimentary tract stasis. * No response to ketosis treatments.

* Normal glucose level.

4- Hypocalcaemia : *No blindness. * Normal glucose level.

* Low calcium level below 4 mg/dl. * Good response to calcium therapy.

5- Hypomagnesaemia: *No blindness – no acetone a dour.

* Sever or clear hyperesthesia. * Usually in 1-2 months after calving.

* Low magnesium level below 1 mg /dl

6- Rabies: * History of biting *Ascending paralysis *Death with mania.

7- Lesteriosis: * The nervous signs are prolonged (not in episodes).

Treatment:- "mainly directed toward elevation of the glucose level"

1- Intravenous administration of 500 ml 40-50% dextrose for cow.

2- Oral administration of glucose after closure of esophageal groove by vasopressin to prevent passing of glucose to the abomasum.

3- Oral glucose forming substances e.g.:

a- Sodium propionate or lactate 120 - 240 gm / day. b- Starch 30 gm .

4- Synthesis of glucose from non – carbohydrates origin by physiological process of gluconeogenesis using some hormones as ACTH. Corticosteroids and glucocorticordes [Dexamethzaone 5-20 mg per head or 10-30 mg Beta – methazone / head for ketotic cow].

5- Cobalt may be useful for ruminal motility and carbohydrates metabolism.

6- Propylene glycol 125-250 gm orally given twice daily for ketotic cows with mineral mixture containing cobalt.

7- Glycerol 120 ml 50% solution [i.e. 120 ml glycerol + 120 ml water] twice daily.

Control:- 1- Providing of food rich in glucose as ground maize

which rich in glucose (alpha polymerized glucose these substance not fermented in the rumen and pass to the intestine and absorbed there)

2- Avoiding of starvation and over fattening at calving.

3- An adequate caloric intake should be ensured in the early part of lactation and especially after treatment.

4- Avoiding the conditions that leading to secondary ketosis as over feeding on diet rich in protein (ketogenic substance) or acidosis ……etc.

5- Good calculated feeding system: (4 weeks prior to calving).

(A)- At calving time it should fed on maintenance ration which is (1kg/ day concentrate and this amount is increased gradually to 5 kg/ day concentrate at the time of calving).

(B)-At lactation the ration is gradually increased as the production increased 3 kg hay / 100 kg body weight + 1kg grain /3 kg milk produced.

N. B 1kg hay = 3 kg silage.

(C)-Protein in the ration should not exceed 16% up to 18%.

6- Ensure that the ration contains adequate amount of cobalt, phosphorus and iodine. 7-Prophylactic feeding of sodium propionate 110 gm daily for 6 weeks commencing the parturition may reduce the incidence of clinical ketosis. 8- Propylene glycol 350 ml daily for 10 days after calving.

9- Monensin (anti–coccidial drugs) as growth promoter leading to increase the ratio of propionate to acetate and gives good results in preventing the disease, but it found that it may lead to depression of the milk fat [25 mg monensin daily in grain feed mix].

9-Downer Cow Syndrome

Definition:- One of major problems in dairy cattle practice is the ,so called downer cow syndrome in which aged animal goes down most commonly during pregnancy and in pere-parturient period. Characterized clinically by prolonged recumbence and at necropsy we found necrosis or injuries of the muscle of the limbs, fatty infiltration, myocarditis and degeneration of the liver.

Etiology:-

I- Metabolic disorders and nutritional deficiency.

II- Non – infectious medical diseases.

III- Infectious diseases

IV- Surgical and obstetrical causes of recumbence.

I- Metabolic disorders and nutritional deficiency:-

A- Metabolic disorders :

1- Disturbance in calcium metabolism → parturient paresis

2-Disturbance in phosphorus metabolism → post–parturient haemoglobinuria.

3- Disturbance in Magnesium metabolism → lactation tetany.

4- Disturbance in glucose metabolism → ketosis or acetonemia.

5- Disturbance in fat metabolism → fat cow syndrome.

B- Nutritional deficiency diseases:

1- Calcium, phosphorus, and vitamin – D deficiency in osteomalicia (Milk lameness).

2- Traces elements deficiencies and disturbance in miner electrolytes:

a- Copper in → falling disease in cattle -

b- Cobalt in → bovine ketosis.

c- Potassium in → Hypokalemia particularly in what so–called." ***Creeper Downer Cow"*** in which the animal is alert and crawl but unable to rise.

d- Protein deficiency in → hypoproteinamia particularly if accompanied by unspecific digestive disturbances.

II- Non – infectious medical diseases-:

1- Sever cases of rumen acidosis (due release of histamine).

2-Liver cirrhosis (due to impairment of liver function especially the detoxification and in activation of steroid hormones particularly estrogen).

3- Liver abscesses (due to hypo – or hyperglycemia).

4- Late stage of pneumonia (due to hypoxia).

5- Late stage of pyelonephritis (due to uremia).

6- Acute circulatory crisis (due to uremia).

7- Traumatic pericarditis (due to generalized toxaemia).

8-Encephalopathy (encephalitis, meningitis, or encephalomalacia).

9- Sever starvation (due to hypoproteinaemia or hypoglycemia).

10- Sever thirst (due to haemoconconteration and dehydration).

11- Leukemia.   12- Sun struck (over heating).

III- Infectious diseases:-

* Generally the last stage of any acute febrile disease may lead to prolonged recumbence for example:-

1- Anthrax. 2- Malignant edema (clostridium spp.)

3- Mucosal disease (MD). 4- Black leg (clostridium spp.).

5- Acute mastitis (due toxemia).

6- Generalized TB (due to tuberculosis meningitis).

7- Last stage of tetanus (due to generalized tonic spasm).

IV- Surgical and Obstetrical causes for recumbence:

* Generally injuries of the locomotor system, musclo–skeletal system (nerves, muscles, joints and bones) can causes in some cases recumbence:

1- Bone : → broken bones (specially vertebral column and pelvic).

2- Joints → arthritis (especially knee or hock joints).

3- Muscles → ischemia, necrosis or muscles or rheumatism.

4- Nerves → pressure on the nerves [especially obturator nerve which pass through the foramen of the pelvic bone] especially in case of too large fetus.

5- Nerves injuries due to paresis or paralysis.

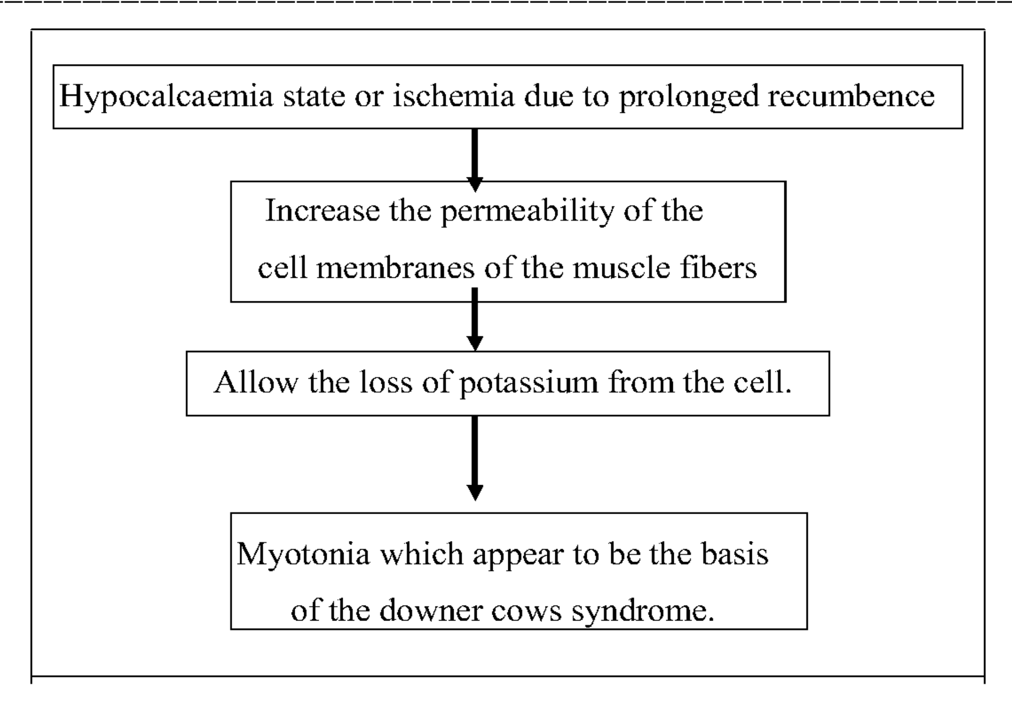
6- Acute endometritis (due to generalized toxemia).

7- Uterine torsion (due to rupture of uterine artery).

8- Uterine rupture (due to diffuse peritonitis).

N.B: 1- Experimentally enforced recumbence for 6, 9 or 12 hours with one hind leg positioned under the body will result in downer cow syndrome.

2- One theory as the following diagram:-



-This view is supported by observed low level of serum and muscle potassium level.

3- Some text books, mentioned that the downer cow syndrome is a condition occurs in cattle usually following milk fever when the cow still in prolonged recumbence even after two successive treatment with calcium borogulconate . and in necropsy finding there is traumatic injuries to limbs muscles and nerves, and ischemic necrosis of limb muscles.

4-Myocarditis due to repeated injection of calcium borogluconate and fatty liver infiltration and degeneration.

5-Etiology not clear but downer cow syndrome may consider as a complication of parturient paresis.

6-Traumatic injuries to nerves of limbs are present in 25% or more of downer cows, sciatic nerve and obturator nerve are vulnerable to injury by pressure from oversized calf before or during parturition.

7-Overlong delay in the treatment of milk fever and prolonged recumbence (more than 4-6 hours) is considered as important cause of downer cow syndrome, due to ischemic necrosis of limb muscles and nerves.

8-Serum electrolytes imbalance or defects have been suggested as a cause of prolonged recumbence [downer syndrome] following milk fever e.g.: Hypokalaemia with hypophosphatemia in the "***Creeper downer cow***".

Clinical Finding:-

A- Typical cases:- 1- Affected animal show no signs until go down.

2- Bright and alert and make frequent attempts to raise but unable to completely extending their legs (sternal recumbence).

3- Eat and drink moderately well and defecation and urination normal while respiration is unaffected.

4-Normal body temperature with week and rapid pulse (80-100 beat/min.)

5-The animal which may remain recumbence for 24 hours or after 2 treatment of parturient paresis can be classified as downers.

6-Tachycardia and arrhythmia may occur following intravenous administration of calcium and sudden death may occurre.

7- The animal makes no effort to bear weight with either for legs or hind legs.

B- Atypical case:- 1- The signs may be more marked and include a tendency to lie in lateral recumbence with the head drawn back and this

animal described as : "***Non – alert downers"*.**

2- In still more sever cases; affected animal may show hyperesthesia and sometime tetany of limbs.

3- These sever cases usually don't eat or drink.

4- If no response after 4-7 days, the prognosis is poor. But some cases may remain for 10-14 days and subsequently stood up and recovery.

5- Death may occur within 48-72 hours following the onset of the disease and usually associated with mayocarditis.

Diagnosis :- 1- Case history and clinical sings.

2- Laboratory diagnosis :

a- Mainly depend upon the causative agent of recumbency.

b- Proteinuria and myoglobinuria with increase the level of creatine phosphokinase enzyme (CPK) up to 40.000 Iv / dl (N= 65 I. V / dl) with increase the level of serum (GOT) which is indication of muscular damage from prolonged recumbence and the muscle damage attributed ischemia of muscles.

****P. M lesions:*** 1- Fatty liver and hepatic degeneration

2- Hemorrhage and edema of the skin which are traumatic origin.

3-Muscle necrosis, ischemia and hemorrhage.

4- Fractured hip bone or wound in the pelvic muscles or tissues.

5-Ulceration at the sites of recumbence

Treatment:- A- Nursing care. B- Chemotherapy

A- Nursing care:- 1- Frequently turning of the animal from side to side to avoid tempany, and muscles necrosis which result from prolonged recumbence.

2- Providing the animal with ample bedding, green food and free water, as well as, not left, the cow on slipping ground surface (to make effort to rise).

3- Massaging of limbs may be successful especially for muscles of thigh as type of physiotherapy. 4- Slinging, but usually unsuccessful.

B- Chemotherapy:- * Mainly depend upon the clinical signs, biochemical finding and the suspected or confirmed cause or causes. But the most satisfactory treatment program includes the parenteral injection of:

1- Electrolytes solutions containing calcium, phosphorus, magnesium, copper, selenium, cobalt and potassium.

2- Nutrient preparation: containing dextrose and amino acids.

3- Polyvitamins preparations containing vitamins A, E, D, B, (thiamin) and B12 (cyanocobalamine).

4- Anabolic hormones may be useful such as steroid gonadal hormones or other anti-inflammatory hormones like corticosteroids or cortisone derivatives particularly if toxemia suspected [as:- Dexamethazen – Beta methazone – Voltarin –…….etc].

5- Fluid therapy by oral or parenteral routs is indicated for cows which not be drinking a normal mount of water.

Control and prevention: - Mainly related to the suspected causes.

10-Fat Cow Syndrome

Definition:-

- It's sporadic highly fatal (100%) metabolic neurotic disease of the pregnant beef cattle especially in late stage of pregnancy when the nutrient intake is decreased in cattle which were previously well fed and in good condition.

Etiology:-

1- Mobilization of excessive quantities of fat from body depots [specially subcutaneous fat] to the liver or may be to the kidney and muscles due to deprivation of feed in fat or beef pregnant cattle especially that which bearing twins.

2- Sever deficiency of energy in the late stage of pregnancy or in the early period of lactation also lead to such syndrome.

3- Fat cow syndrome considered as exaggerated form of ketosis in the pregnant beef obese cows.

Incidence ,Predisposing Factors and Occurrence:-

1- Its most common in: - * Pregnant beef obese cattle.

* In late pregnancy [last 2 months]. * In cows carry twins

* In the first – calf – heifers than older cows.

2- Good feeding in the first period of pregnancy then the animal subjected to sever nutritional stress as long period of starvation. Especially in the last 2 months of pregnancy acts as a predisposing factor to fat cow syndrome.

3- More common in high producing cow. When the diet intake not to

meet the energy demand leading to energy defect which stimulate the mobilization of fat from the fat depots in the body to the liver causing fatty infiltration of liver.

4- Morbidity is very low (1-3%) while the mortality up to 100%.

5- It's more common in cattle that have more than 20% of the liver fatty infiltrated before the occurrence of the disease.

6- Cattle of long dry period also have the tendency to become obese and develop the fat cow syndrome.

4-It may be occurred immediately after calving.

8- Abnormal change in the diet of pregnant cows especially in the late stage of pregnancy from silage to straw may considered as predisposing factor, due to disturbance in the energy system.

Pathogenesis:-

1-Normally fat is present in the liver in appreciable amount sup to 5% of the liver and these ratio is increased in the late stage of pregnancy, in 2 weeks before calving and 1 week after calving up to 20% (Why…????) then declined until the normal level up to 5% at 26 weeks after calving (i.e. about 7 months).

2- The following diagram show what happen in case of fat cow syndrome

3- Triglyceride is the most fat that accumulated in the liver.

4- Fatty infiltration of the muscle also occurred which leading to muscular weakness and recumbence.5- Degree of the disease may be mild, moderate and sever depend upon amount of fat that infiltrated in the liver.

Shortage in the feeding system or inability of the cow to take adequate amount of the food especially in the critical period of the pregnancy or early lactation

↓

Decrease the energy intake

↓

Increase the fat catabolism for production of glucose and energy by gluconeogenesis

↓

Mobilization of excessive amount of free fatty acids

↓

- Increase the liver lipogenesis
- Accumulation of the lipids in the large hepatocyte
- Depletion of liver glycogen
- Inadequate transport of lipoprotein from the liver

↓

Fatty infiltration of the liver (Fat cow syndrome

Pathogenesis Of Fat Cow Syndrome

Clinical Findings:-

1- The disease usually occurs in fat cows and at the late stage of pregnancy or few days after parturition.

2- Affected cows are depressed for 10-14 days.

3- Sudden complete anorexia.

4- Sternal recumbence.

5- Transitory period of restlessness, excitement, in-coordination, stumbling gait and aggressiveness.

6- Dry and cracked muzzle which may be peel off.

7- Scanty feces and firm terminally become soft diarrhea and yellow orange, but still small in volume.

8- Tachycardia and increased pulse rate and respiratory rate.

9- Respiratory grunt and clear nasal discharge.

10- The cow become comatose and dies quietly after the course of the disease about 10- 14 days.

Diagnosis:-

A- Case history and clinical symptoms.

B- Laboratory diagnosis:-

1- Firstly hypoglycemia end by hyperglycemia in hepatic failure [N= 40- 60 mg%].

2- Ketonemia [N = up to 10 mg/dl] and ketonuria [N= up to 70mg%].

3- Increase of the liver function.

a- Excessive billirubin.

b- High enzyme → Got, GPT and ALP

4- Increase of Beta hydroxyl butyric acid [ketone body].

5- Decreased insulin, albumin and cholesterol.

6- Proteinuria due to fatty infiltration of the kidney.

7- Liver biopsy to determine the severity of the disease by 2 methods.

a- Biochemical determination of triglyceride

Normal level of neutral fat is 150 mg%.

b- Detection of the lipid contents of the liver physiologically.

[N= Not more than 20%].

Treatment:-

1- In generally, cows which are totally anorexic for 3 days or more or recumbent the treatment is ineffective and the animal will die.

2- There is a slight response (transitory) to parenteral treatment with glucose, glucocorticoids, cobalt and vit – B12.

3- Intensive therapy should be directed to correct the pathophysiological effects of the disease and fatty liver by:

(A)- Continuous I/ V injection of glucose electrolyte solution (Dextrose 5, 10, 25, 40 % or dextrose + saline).

(B)- Interaruminal administration of ruminal juice (5-10 liters) from healthy cow to stimulate the appetite of affected cow.

(C)-Oral administration of propylene glycol or glycerol to promote the glucose metabolism.

(D)-s/c injection of insulin (zinc protamine preparation) to promote the peripheral utilization of administrated glucose, the dose is 200-300 Iu. twice daily.

(E)-s/ c administration of choline chloride in sever cases 25 gm every 4 hours.

(F)-water and balanced electrolytes (10-30 liters) can be administrated intra ruminal.

N. B :

*Injection of insulin and glucose in case of fatty cow syndrome leads to decrease the mobilization of lipids from the fat depots to the liver and help in the rapid removing of triglyceride from the circulation.

Control:-

Because of the large economic losses associated with the Fat cow syndrome so, the main items of control are:-

1- Prevent the pregnant beef cattle from becoming fat during the last trimester of pregnancy.

2- Apply metabolic profile test as mean of assessment of the energy status by estimation of both glucose and Beta hydroxyl butyric acid periodically especially in the late stage of pregnancy.

11-Deficiency of:-

-Vitamin E and Selenium

-Cobalt

-Iron

-Iodine

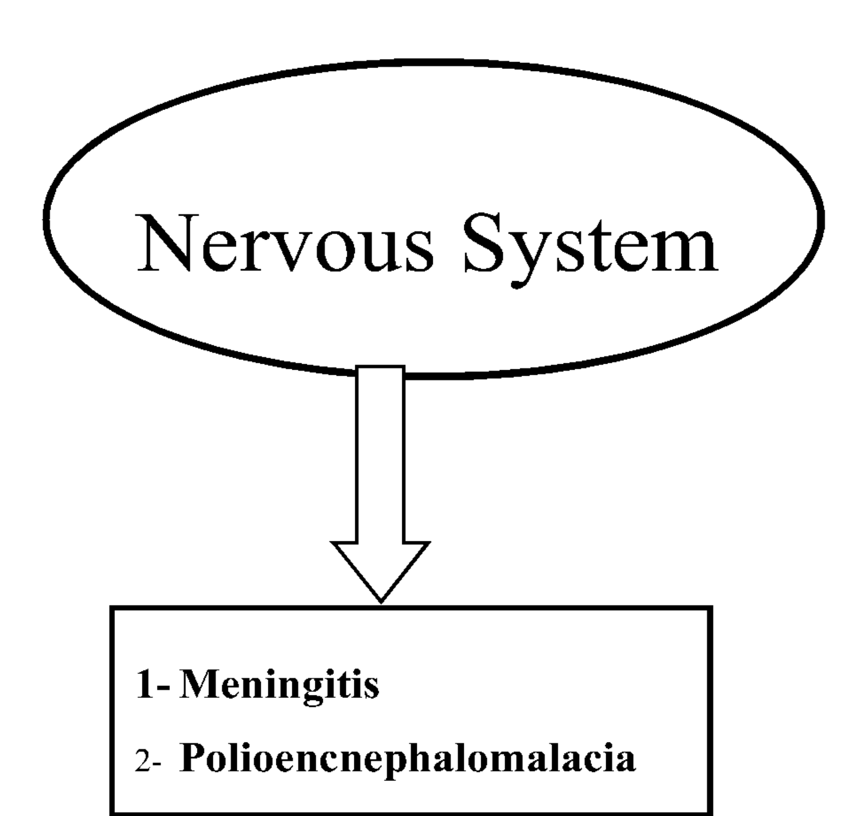
-Zinc

-Vitamin –D

-Vitamin –A

(As mentioned before in the Part (I) Small Ruminant Medicine Sheep And Goat).

Chapter No.(6)



1-Meningitis

Definition:- Inflammation of the meanings and it is usually caused by bacterial infection and clinically characterized by fever, rigidity in the muscles and hyperesthesia

Etiology:- 1-Staphylococcus aureus 2-Streptococcal infection 3-Coliform septicemia

Pathogenesis:- 1-Inflammation of the meninges leads to swelling of the meninges and it leads to interference with the blood supply to the brain and spinal cord which may be followed by cerebral anoxia .

2-Also there is inflammation around the nerve trunk which passes across the subarrachnoid spaces of the meninges

3-The signs produced by meningitis are a combination of the result of irritation of both central and peripheral nervous system which mainly are muscular tremors and convulsions.

4- As the main cause of the meningitis is usually bacterial infection, so the sings of bacteremia or toxemia may be associated with meningitis.

5-Meninigitis also lead to defect in the drainage system of the cerebrospinal fluids (CSF) that may lead to appearance of the symptoms of increased intra-cranial pressure together with the sings of meningitis.

Clinical Symptoms:-

1-Fever with signs of toxemia 2-Drowsness and coma

3-Trismus, Opisthotonus and rigidity of the neck and back

4-Muscular tremors, convulsion and paddling movements

5-Cutaneous hyperesthesia is present in varying degree, even light touch of the skin may cause sever pain and excitation in some cases

6-Blindness is common in cerebral meningitis, but not a constant clinical finding

7-Pupillary light reflex is usually much slower than normal.

Diagnosis:- (A)- Case history and clinical sings

(B)-Clinical pathology: - 1-Leukocytosis

2-Presenc of bacteria in bacterial culture of CSF.

(C)- PM. Lesions:- Hyperemia and hemorrhage in the meninges.

Treatment:-

1-Because of the main cause of meningitis is usually bacterial infection so the main points in the treatment are parental administration of antibiotics daily for several days:-

2-Administration of the antibiotics in the lumbosacral spaces has been recommended but the method of administration is very difficult to be applied daily.

3-If there is no response after 3-5 days of the treatment, so the prognosis is unfavorable.

4-The best antibiotics that can be used in the treatment of minigtits are

-Penicillin -Tetracycline -Chloramphenicol -Aminoglycosoides preparations

2-Polioencephalomalacia

Definition:- It is also known as cerecbrocortical necrosis ,it is a nutritional / metabolic disease affecting the ruminant ,and it is means that softening of the gray matter of the nervous tissues.

Etiology & Pathogenesis:- 1-The main cause of polioencephalomalacia is deficiency of thiamin or disturbances in the metabolism of thiamin.

2-Thaimin is a part of the enzyme of thiamin pyrophosphate which play an important role in the carbohydrate and amino acids metabolism.

3-The enzyme of thiamin pyrophosphate play an important role in the glucose metabolism so deficiency of the thiamin leads to disturbances in the function of thiamin pyrophosphate enzyme and disturbances in the carbohydrate metabolism ,so the nerve tissue which depend on the glucose will be affected by the thiamin deficiency.

4-Deficincy of thiamin leads to edema and death in the nerve cells and the clinical sings of the disease will be devolved from the diffuse cerebral and cortical necrosis and edema that occurred.

5-Also excessive feeding of molasses or amporlium drugs may also leads to polioencephalomalacia with unknown interpretation.

6-Also feeding on moldy food and sever lactic acidosis may act s a predisposing factors of polioencphalomalacia.

Clinical Signs:- 1-Subclinical deficiency of thiamin may be associated with poor growth rate

2- Nervous manifestation in the form of: - A-Walking in circling form.

B-Staggering in gait C-Tremors and convulsions

D-Untreated cases will be die after 48-72 hours after the appearance of the clinical symptoms. E-Nystagmus or blindness.

3-Weakness of all four limbs is accompanied by dullness, ataxia and head pressing. 4-The hind legs are more affected than fore legs.

5-The clinical signs may progress slowly and stabilized for long periods.

Diagnosis:- 1-Case history and clinical symptoms

2- Low blood level of thiamin.

3-***P.M. Lesions*** reveal softness and edema in the brain tissues with yellowish or grayish yellow discoloration of the brain tissues.

Treatment & Control:- 1-Treatment and prevention of the disease depend upon maintenance of the level of thiamin within the normal requirement of the animal. 2-Using of thiamin hydrochloride in the treatment 3-Parenteral dexamethason 0.1mg/kg. Bwt. May be used in sever cases to prevent brain edema.

4-Intravenous administration of hyperosmotic manitol in a dose of 1.5 gm/ kg Bwt. 5-Diazepam or other type of anticonvulsant preparation may be used 6-Any underplaying problems that may have predisposed to polioencephalomalacia, such as grain engorgement (impaction) should be identified and treated and its complications as dehydration and metabolic acidosis. 7-Avoid feeding of high amount of molasses and moldy food.

8-Addition of thiamin on the ration or addition of brewer's yeast.

Part (III)

Camel Medicine

1-Clincal Examination Of The Camel

How to examine a camel?

I)-History

A)-About the animal:-

1-Age and sex of the animal

2-Mother – animal relationship (if the animal is a calf)

3-What the diseases had it in the past?

4-Last feeding, drinking, urination and defecation

5-Has it been treated by any medications previously?

B)-About the herd:-

1-Are there any other diseased animal in the herd(if the infamous diseases are suspected

2-Dead animal and its PM. Lesions

C)-About the surrounding:-

1-Where the animal has been kept?

2- Where the animal grazing?

3-Sources of the water and food

(II)-Physical examination of the camel

(A)-Inspection:-

(1)-Behavior and appearance of the camel:-

A-Unusual behavior as:-

-Laying down and getting upon frequently

-Lack of appetite

-General weakness and emaciation

-Scratching of the body

B- Difficult in the breathing

C-Coughing (cough can be induced by offering some water or food to the animal on the ground, so that the animal lower its head and neck and may cough).

D-Unusual posture and walking.

E-Swelling of the joints.

F-Abnormal shaped hump.

(2)-Skin:-

-Unusual appearance of the skin and hair.

-Presence of wounds or ulcers.

-Presence of the external parasites.

-Presence of edema or swelling.

(3)-Mouth, nose and ears:-

-Color of mucous membrane of the mouth or nostrils is normally pink color.

-Presence of nasal discharges or salivation and the nature of these discharges (unilateral, bilateral or serous……..etc.)

-Discharges from one or both ears.

-Abnormal smell from the animal

(4)-Eyes:-

-Presence of tears or any other discharges. -Swelling of the eye lids.

-Color of the conjunctival mucous membrane (Normal is pink in color)

(5)Feces and urine :-

-Normal feces is hard, round, oval and green. -Presence of worms in the feces

-Color of the skin inside the anal opening

-Presence of any discharges from the vagina.

-Presence of diarrhea or constipation or straining during urination or defecation.

-Presence of any abnormal things in the urine or feces as in case of presence of blood.

(III)-Examination of Body Temperature, Pulse, Respiration and Rumination:-

1-Body temperature in camel is taken racially.

2-Normal body temperature is (average 37.5 C).

3-The normal temperature is greatly varied in the day within 6C (it is considered as the most common problem in taking the body temperature of the camel)i.e. ranged from 34.2-40.7C and it is lowered in the morning and elevated in the evening and the variation is greatly increased if the animal has not been watered for long period.

4-The normal pulse rate in camel is 30-50 /minutes.

5-The pulse can be taken by auscultation of the heart.

6-Normal respiration in the camel is 5-12/min. in cold weather, 8-24 /

min. in hot weather and 14-16 /min. in the young animal up to three months in age.

7-The normal respiratory type in camel is abdominal

8-Check the activity of the rumen with the stethoscope on the left side behind the rib cage for 3-4 minutes to hear the ruminal movement

9-Normal ruminal movement in camel is 3-4 times per minute if the camel ruminating.

(IV)- Diagnosis & Treatment of Camel Diseases:-

(A)- Difficulties in diagnosis of camel diseases:-

1-Some different diseases may show the same clinical symptoms.

2-The animal may suffer from more than one disease, so some clinical symptoms of the diseases may be masked or reinforced by the other symptoms of the other disease.

3-Because of the camel is very placid animal, so it may not show any sighs of the illness.

(B)-Common signs of he diseases in camel:-

1-Weakness

2-Tirdness

3-Dullness

4-Laying down at unusual time

5-Lack of appetite

6-Rapid heart rate and abnormal respiration

7-Fever which characterized by the following signs:-

-High body temperature

-Fast pulse and sweating

-Standing with head down and still dull with half closed eyes

-Camel doesn't ruminate

8- Signs of the pain as the following:-

-The neck is erected and stiff

-Sitting uneasily

-Grunting when breathing or ruminating

- Sometimes the neck is raising and lowering quickly

NB.:-

___Grinding on the teeth with foam in the mouth is common in camel after eating a salt diet or in the male camel in the rutting season.

(C)-Tentative Diagnosis of camel diseases:-

1-Often you will not be sure exactly what the disease is so in such condition you should decide what the disease probably is and the treatment of it ,this case is called (tentative diagnosis).

2-If the treatment works, continue in using of it but if there is no response of the given treatment according to your tentative diagnosis it should be changed to another remedy.

3- In the practice this method ids often the only one possible method especially for hard to diagnosis problems as skin problems, infectious diseases, diarrhea, cough and pneumonia.

2-Most Common Diseases Of Camel

Disease	Common Causes
(I). Skin Diseases	
1-Mange	-Parasites (sarcoptic scabie var can camelei)
2-Ring worm	-Trichophyton and Microsporium
3-Pox	-Orthopox virus
4-Orf.	- Parapox virus
5-Abscess in lymph node	-Coryn. in adult or staph. or strept. in calf camel
6-Snake bite, wound, burn, saddle sore or chest pad abscess or fleas infestation	-According to the history and signs of each case
(II)-Nose and Lungs	
1-Cough and pneumonia	-lung worms
	-Bacterial infection (hemorrhagic septicemia)
	-Choke
	-Viral infection
2- Nasal Bots	-Cephalopina titilator
3-Lung worms	-Dictycaulus filarail and viviparous
(III)- Stomach and Intestine	
1-Diarrhea	-Internal parasites
	-Drinking of too much milk in calf camel
	-Bacterial or viral infection
	-Sudden changes in diet
	-Plante poisning
	-Stress
2-Constipation	-Lack of fibers in the diet
	-Feeding too much pelleted diet or grains
	-Feeding on low quality roughage
3-Haemorrhagic enteritis	-Closteridial infection(C. perfringen)
4-Bloat	-Overfeeding on tymapnogenic food
	-Drinking large amount of water after grazing
5-Indigestion	-Sudden changed in the diet

	-Stress
	-Eating of alot of concentrates, grains or flour
(IV)-Head,Legs,feet,Nech &Tail	
1-Broken jaw	-Trauma
2-Goiter	-Iodin deficiecny
3-Blister in the mouth	-Infection or injuries in the mouth
	-Vitamin B deficiency
4-Eye problems	-Foreign body or injuries
	-Eye worms
	-Vitamin A deficiency
	-Conjuncitivitis
5-Ear problems	-Trauma
	-Entering the water inside the ear during bath
	-Mites or ticks inside the ear
	-Bacterial infection
6-Broken bone	-Trauma
	-Accident
	-Old aged animal
	-Over work
7-Arthritis	-Bacterial infection
8-Myopathy	-Over-excretion
	-Lack of movement followed by vigorous work
	-Plant poisoning
	-Genetic factors
9-Sore feet	-Sole injuries
10-Tail gangrene	-Neglected old wound in the tail
(V)-Infectious Diseases:-	
1-Trypansomaisis	-Trypansoma evansi
2-Hemorrhagic septicemia	-Pasteurella multocida
3-Anthrax	-Bacillus antharcis
4-Black quarter	-Closterdial chauvoei

5-Rabies	-Virus transmitted by biting from infected animal
6-Rift valley fever	-Virus transmitted by the insect
(VI)- Non - Infectious diseases	
1-Red urine	-Infectious diseases of the kidney
	-Urolethiasis
	-Internal haemorrhage
	-Plant poisoning
	-Wound in the scrotum
2-Sun stroke	-Over heat or deficiency in mineral or vitamins
3-Allergy	-Certain vaccine (specially that are oily)
	-Certain type of food
	-Injection of some drugs
	-Insect bites
4-Pica	-Lack of salt in the diet
	-Vitamins deficiency
	-Lack of certain mineral as calcium
	-Internal parasites
5-Downer camel	-General weakness
	-Muscular injuries
	-Nerve damage
	-Exhaustion
	-Snak bit
	-Broken bone
	-Tick paralysis
	-Haemorrhagic diseases
6-Dry coat	-Lack of salt in the diet
	-Over work and improper rest
	-External parasites
7-Odema	
-In the udder	-Internal parasites- Mastitis- Trypansomaiasis
-In the face	-Pox or Orf . Infection
-Around the wound	-Bites

-In the penis	-Trypansomiasis
-In the legs	-Bites or arthritis if swelling in the joints area
(VII)-Calf Camel problems:-	
	-Navel ill
	-Salmonellosis
	-Colibacillosis
	Diarrhea

3- Cough, Colds and Pneumonia

Etiology:-

1-Coughing and runny nose (colds)or pneumonia in camel can be caused by may different things ,including worms, and other parasites ,viruses and bacterial infection

2-Virus and bacteria infection as:-Influenza virus, hemorrhagic septicemia

3- An illness may begin as a viral infection and then, later it turned into bacterial infection especially if the animal not has enough rest in the first few days of the infection

4-Parasites as lung worms (See most common diseases of the camel)

5-Aspiration of medication during oral administration

6-Pneumoina is rare in camel but it is very hard to be diagnosed

7-Diseaes of the nose and lungs can be transmitted in different ways, can be transmitted from diseased animal to the healthy one by coughing and sneezing specially when they are closed together during watering or in the pen at night. 8-Parasites can be transmitted through the contaminated pasture.

Clinical Symptoms:-

1-Coughing and sneezing 2-Difficult in breathing or drinking

3-Over breathe more than 15 per minutes

4-dilated nostrils with mouth breathe in some cases (signs of dyspnoea)

5-Drooling from the mouth with runny nose (nasal discharges)

6-Inapetance to anorexia. 7-Fever shallow rapid respiration

Treatment:-

1-Antibiotics (usually injected) such as oxytetracycline for about 5-7 days in case of bacteria infection.

2-Anthelmints in case of lung worms or internal parasites as piprazin citrate (3gm.710 kg.Bwt. orally).

3-Decongestant (for cleaning of the nasal discharges) as ephedrine sulfate 1% 2drops in each nostril every 3 hours

4- Isolation of the diseases animal in good ventilated area with complete rest.

4-Diarrhea

Etiology:- 1-Diarrhoea is common in calf camel and causes death of many of them.

2-Diarrhea usually occurred in the adult animal in the beginning of the rainy season

3-See table of the common diseases of the camel (stomach and intestine).

Clinical Symptoms:-

1-Change in the color and consistency of the faece.

2-General weakness with loss of the condition

3-Fever if the cause is infectious (bacterial or viral).

4-Swollen belly 5-Sunked eye with the other signs of dehydration.

6-Stop suckling in case of calf camel.

Prevention And Treatment:-

1-Isolation of the diseased animal away from the healthy one.

2-Moveing the animal from the pasture which may be contaminated with faeces 3-Keep the animal away from the poisoning plant.

4-Control the milk intake in the calf camel (avoid over-feeding).

5-Treat with oral rehydration fluids

6-Mix 200 ml. of kaolin or 100 gm. of tannic acid with about 1 lire of water and use it for the adult animal daily until recovery while in the calf camel we can use the half of this dose.

7-Parenteral administration of long acting antibiotics.

8-Oral administration of antidiarrheal drugs.

5-Bloat

Etiology:-.

1-Bloat (build up of gases in the rumen) is not a common disease in the camel perhaps because of they are able to vomit

2-See the table of common disease of camel (bloat).

Clinical Symptoms:-

1-The belly is swollen on the camel `s left side (the side where the rumen is

present). 2-Mild signs of colic

3-The animal stop eating and drinking

4-The animal rolls on the ground on the right and left side but on the back.

5-The animal may die within one hour in very sever untreated cases.

Prevention And Treatment:-

1-Aviod feeding on the tympanogenic diet

2- Not allow to the camel to drink large amount of the water especially after grazing .

3-Drenching of about 300-500 ml. of anti frothing agent as (Bloatremady. Dimethicon, Gase remedy……etc.).

4-Intreavenous administration of 10-15 liters of Ringer's lactate for 3 days

5-Drenching mixture of 1kg. Of salt, 1 kg. Of magnesium sulfate, and 1-2 liters of the cooking oil and allow the animal to walk about 5oo meters after giving the drench.

6- S/C administration of Neostagmine (1mg. / 50 kg. Bwt.) daily for three days.

7-Using of the stomach tube to escape the gases from the rumen or for administration of the drugs intraruminal especially in acute or sever cases

8-Using of trocar and canula (on the left side of the abdomen 10cm.or about one hand width behind the last rib, and 7.5 cm. below the ridge of the animal's side that formed by the bones in the spin.

6-Indigestion

-Camel that given fed on large amount of concentrates or there is sudden changes in the diet system may be suffer from indigestion

-See the table of most common disease of the camel (Indigestion).

Clinical Symptoms:-

1-The animal vomits violently 2-The vomit is acidic 3-No rumination

3-Presence of foul and acidic smell from the mouth. 5-Weakness

Treatment:- 1- Dissolve about 1 kg. Of sodium bicarbonate in water and drench on the first day of the disease followed by 100 g. per day for 3 days (if the disease is mild this treatment is sufficient)

2-Injection of hyoscine derivatives to calm the gut (Buscopan about 20-30 ml-IM.)

3-Drenching of about 250-500 g. of magnesium sulfate in about 5 liters of water

4-Adminsteration of preparation of anti-indigestion, as Vapkodigest, Bykodigest, Stomaton…..etc.

5-In sever case we can administrate solution of sodium bicarbonate (60-120g.in concentration of 1.5-3%) once daily for 1-2 days

6-Adminsterationof saline solution (0.9%) intravenous about 10-15 liters per day for about three days

7-Application of ruminal transplantation from freshly slaughtered animal or from the healthy on by the stomach tube.

Chapter IV

General Systemic and Poisoning diseases

1-Dehydration **2-Edema**

3-Hypothermia **4-Sudden death**

5-Anemia **6-Stress**

7-Selenosis (selenium toxicity)

8-Copper toxicity **9-Pain**

10-Snak bite

11-Organophosphorous poisoning

1-Dehydration

Definition:- It is one of the most important disturbances in the water and electrolytes balance in which there is more fluid lose from the body than that absorbed that leading to reduction of the circulating volume of the blood and dehydration of the body tissues(loss of the body fluids).

Etiology:- There are two major causes of dehydration

(A)- ***Failure in the water intake as in case of***:-

-Deprivation of water -Thirst due to toxemia

-Inability to drink as in case of choke

(B)-***Excessive water loss as in case of:-***

-Vomiting -Hemorrhage -Copious sweating

-Diarrhea -Polyurea -Excessive salivation

-Extensive skin wound as in case of burn

(C)- ***Other causes as in case of:-***

-Carbohydrate engorgement (Impaction) -Diffuse peritonitis

-Intestinal obstruction -Abomasal Displacement

-Dilatation or torsion of the abomasum

Adaptation of the animal against dehydration:-

-The ability of the animal to survive for long period without water in the hot climatic conditions deepened on:-

A-Degree of the insulation B-Amount of the extra cellular fluids

C-Amount of the water reserve in the rumen in the ruminants animals

D-The ability of the animal to adjust the electrolytes concentrations in several fluids locations

E-The ability of the kidney to conserve water and the ability to maintain the circulation with lower plasma volume.

Pathogenesis:-

(1)- There are two factors involved in the pathogenesis of dehydration

(A)-Depression of the body tissues fluids levels with resulting of the reduction of the tissues metabolism

(B)-Anhydration (reduction of the fluid contents of the blood)

(2)-Etiology of the dehydration leading to negative water balance and the ***initial response to the negative water balances*** is withdrawing of the fluids from the tissues to maintain the normal circulating blood volume and this occurred by:-

(I)-The fluid is drained primary from:- → Intravascular compartment
→ Interstitial spaces

And the most essential organs that included in this process are CNS, heart and skeletal muscles

(II)-Loss of the interstitial fluids and cellular fluids leading to:-

-Loss of the skin elasticity -Dryness of the skin and mucous membrane

-Enophthalmia (reduction and retraction of the eye ball) due to reduction in the postorbital fat deposits.

(III)-Loss of the circulating blood volume (reduction of the fluid contents of the blood) leads to acidosis or toxemia according to the main cause of the

dehydration then leads to mental depression of the dehydrated animals.

-Secondary response to the negative water balance :-

(I)- Reduction of the water contents of the blood which may lead to

A-Reduction of the volume of the circulating blood (oligemia)

B-Increase the concentrations of the blood contents (haemoconcentration)

(II)-Haemococnetration leads to:-

A-Increase the viscosity of the blood which impaired the blood flow causing peripheral circulating failure.

B-Decrease the renal blood flow which manifested by oliguria and production of concentrated urine

(3)-In case of deprivation of the water and electrolytes or inability of drinking the water as in case of esophageal obstruction the dehydration in the animal is caused by:-

A-The kidney compensate effectively by decreasing the water output and increase the water reabsorption

B-Preservation of the water by reducing water contents of the feces and increase the intestinal absorption of the water and this leads to dryness of the ruminal contents and producing the dry feces (constipation).

(4)-Dehydration leads to some important effects on the tissue metabolism as increase the catabolism of the fat(breakdown of the fat) ,then the carbohydrate and finally the protein to produce water that resulting from the metabolic process which leading to finally to produce acid metabolites causing acidosis and increase the urea nitrogen of the blood.

Types Of Dehydration:-*Dehydration can be classified according to the degree of loss in the water and electrolytes (sodium) as the following:-*

(A)-Hypertonic dehydration:- -This type of dehydration characterized by loss or deprivation of water with minor loss of sodium (uncommon) as in case of inability of drinking of water as in case of esophageal obstruction .

(B)-Isotonic dehydration:- -This type of dehydration characterized by mild loss of water and sodium within equal manner and there are mild degree of dehydration and hyponatreamia, Examples:- Simple enteritis - Nephrosis - Copious sweating.

(C)-Hypotonic dehydration: - -This type of dehydration characterized by severs loss of water and sodium and there are sever degree of dehydration and hyponatraemia, Examples: - Enterotoxaemia - Salmonellosis.

(II)-Dehydration can be classified according to the degree of the loss of the body fluid as the following table:-

Degree	Loss of body weight %	Sunken eyes	Skin fold test(sec.)	PCV.%	Serum sodium gm./L.	Fluid needed ml/kg.Bwt.
1	4-6	+	-	40-45	70-80	20-30
2	6-8	++	2-4	50	80-90	30-50
3	8-10	+++	6-10	55	90-100	50-80
4	10-12	++++	20-45	60	120	80-120

Clinical Symptoms:-

1-Dryness of the skin and mucosa and loss of the skin elasticity

2-Pale mucous membranes with sunken eye and exophthalmia

3-Wrinkling of the skin 4-Long time of the skin fold test

5-Body temperature initially is above normal level (dehydration hyperthermia) due to insufficient body fluid that needed to maintain the water loss by evaporation while in the late stage of dehydration the body temperature is subnormal (hypothermia) due to sever insufficient of body fluid which leads to muscular weakness and decrease the metabolism that leads to decrease the heat production resulting hyperthermia in the late stage of the dehydration.

6-Specific signs of the specific cause of the dehydration as in case of chock there is difficult of swallowing...........etc.

7-Decrease of animal productivity and general weakness.

Diagnosis:-*(A)- Case history and general clinical symptoms .*

(B)-Skin fold test or capillary refill time test

(C)-Lab. diagnosis (increased PCV, hyponatraemia and acidosis).

Treatments:- 1-Fluid therapy as saline solution and sodium bicarbonate 2-5 % IV. To overcome acidosis

2-Treatemtne the main cause of dehydration as in case of impaction or enteritis or vomiting.............etc.

3-Placing the animal good ventilated place with clean and fresh adequate water supply.

2-Edema

Definition:- Edema in the excessive accumulation of fluid in tissues spaces or body cavities caused by disturbance in the mechanism of fluid interchanges between capillaries , the tissue spaces and lymphatic vessels.

Etiology:- Edema resulted mainly from:-

(A)- Increase the hydrostatic pressure of the blood capillaries

(B)- Decrease the plasma osmotic pressure (hypoproteinemia)

(C)- Obstruction of the lymphatic drainage or flow

(D)- Vascular damage of small vessels or damage of the capillary wall

(A)- Increase the hydrostatic pressure of the blood capillaries:-

as in case of:-

1-Generally in congestive heart failure

2-Locally in portal hypertension due to hepatic fibrosis causing ascitis

3-Locally by compression of mammary vein by large fetus causing mammary or ventral edema in mare or cow in the late stage of pregnancy (cake or boat belly).

(B)- Decrease the plasma osmotic pressure (hypoproteinemia):-

as in case of:-

1-Continued blood or protein (hypoproteinemia) loss especially in the heavy infestation of the blood suckling parasites as in case of

-Strongylus Spp. In equine -Fasciola spp. In the ruminants

-Homonchus spp in all ages of the ruminants especially in goats

2-Renal diseases which cause loss of protein, but nephritic edema occurred rarely as in case of nephrotoxic substances

3-Protein losing gastroenteropathies as in Johne`s disease or Enterocolitis Or Heavy infestation of nematodes specially ostertagia spp. in young cattle

4-Liver damage causing failure of synthesis of protein (plasma protein).

5-Malnutrition with diet low in protein (starvation).

(C)- Obstruction of the lymphatic drainage or flow as in case of :-

1 –Lymphangitis in horses 2-Edematous skin disease in cattle.

3-Congential obstruction of the lymphatic

4-Trauma or inflammation in the lymphatic system which may lead to ascitis or hydrothorax.

5-Edema of the limbs in the immobilized animals due to injuries or illness that mainly caused due to poor lymphatic and /or venous return of the affected limbs

(D)- Vascular damage of small vessels or damage of the capillary wall as in case of:-

1-Toxic vascular damage as in case of :-Anthrax -Gas gangrene -Malignant edema -Viral arteritis . -Intravenous administration of Ivermectin (Ivomec) preparation.

2-Allergic edema, urticaria, or angionueretic edema or photosensitization due to liberation of vasodilators as histamine that leading to sever engorgement of the small vessels and capillaries with blood and rupture leading to edema.

Pathogenesis:-

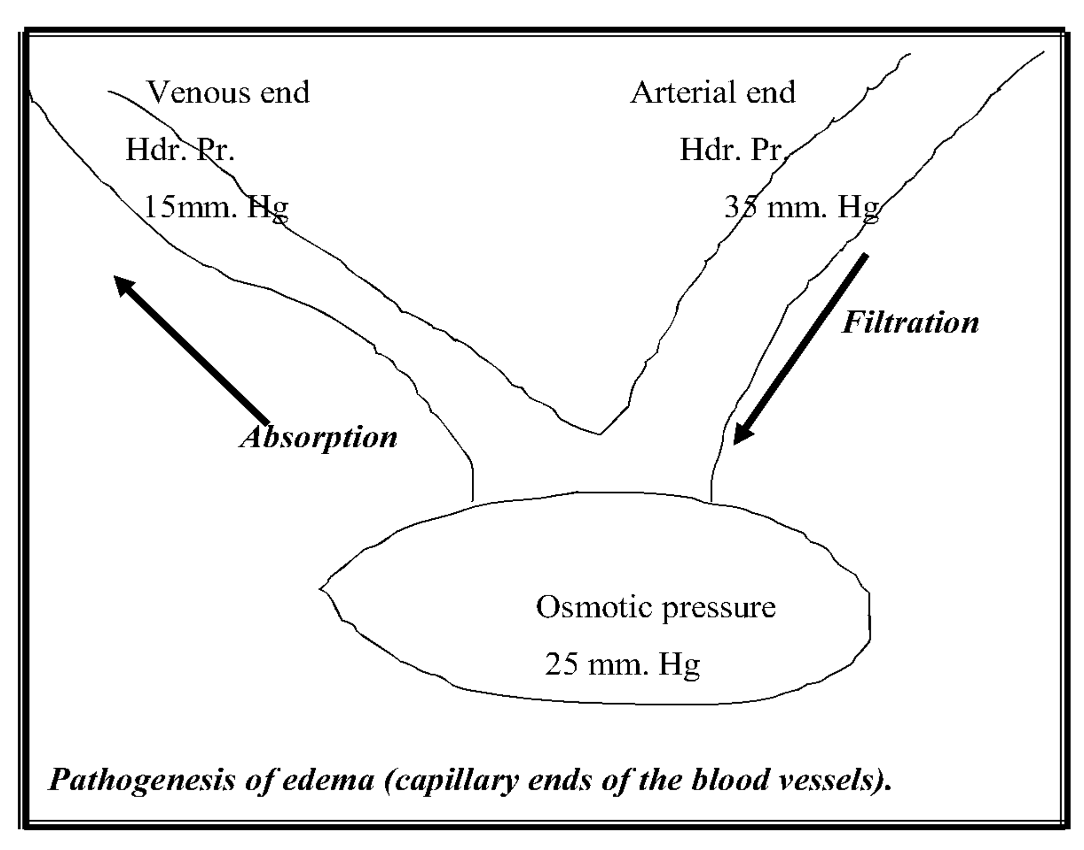
1-At the arterial ends of the blood capillaries:- the hydrostatic pressure of the blood is (35 mm. Hg) sufficient to overcome the osmotic pressure(25 mm. Hg.) that leading to pass the fluid into the tissue by the process what so called "***Infiltration*".**

2-At the venous ends of the capillaries:-the position is reversed as the hydrostatic pressure is 15 mm. Hg. While the osmotic pressure is 25 mm. Hg. so the fluid tends to return to the vascular system by the process what so called "***Absorption*".**

3-The pressure different enter the arterial part and venous part of the blood capillaries and the osmotic pressures, so a small increase of the hydrostatic pressure or decrease in the osmotic pressure leads to failure of the fluid to return to the capillaries (venous return) leading to accumulation of the fluids in the tissue spaces or body cavities causing edema

4-Lymphatices convey the plasma protein that filtrated in the tissue to the circulation, so the lymphatic play an important role in maintenance of the colloidal osmotic pressure of the plasma so any diseases of the lymphatic as inflammation or obstruction may lead to edema.

5-The main type of protein that play an important role in the osmotic pressure of the plasma is albumen so any diseases that associated with hypoproteinemia (hypoalbumenemia) usually associated with edema as in liver or kidney diseases.



Pathogenesis of edema (capillary ends of the blood vessels).

<u>Clinical Symptoms</u>:- Vary according to the location

<u>1-***Anasarca***</u> which means that accumulation of the transudate fluid in the subcutaneous tissue usually in the ventral abdominal part or in the neck.

2-***Ascites*** which means that accumulation of the edematous transudate in the abdominal cavity

<u>3-***Hydrothorax***</u> accumulation of the edematous transudat in the thoracic cavity

<u>4-***Hydropericardium***</u> accumulation of the edematous transudat fluid in the

pericardium sac.

5-***Bottle jaw*** accumulation of the edematous transudate in the intermandubular space especially in the grazing animals as in case of fascioliasis due to lowering of the head during grazing

6-***Local edema*** ***(*** in the horse) specially in the head region as in case of African horse sickness or purpura hemorrhagica, or in the legs in cattle as in case of edematous skin disease due to obstruction of the lymphatic.

7-The edematous swelling is soft, painless and gives pit in pressure.

8-Edema of the pericardium or pleural cavity usually associated with muffled percussion and auscultation with difficult in respiration.

9-***Cerebral edema*** usually associated with nervous sings.

10-***Pulmonary edema*** usually associated with foamy nasal discharges, respiratory distress with loud harsh sound in auscultation.

11-Ascites characterized by thrilling by tactile percussion or fluid sound by auscultation and it can be detected by parasentesis.

12-Other sings of the specific cause as in case of heavy infestation of internal parasites or in case of hypoproteinaemia.

Diagnosis:-

(1)-History and clinical sings

(2)-Ascetic fluid rich in protein (albumin) and free from inflammatory cells

(3)-Hypoproteinemia

(4)-Positive fecal examination tests if parasitism is suspected.

(5)-***Differential diagnosis*:-** edema should be differentiated from other

abnormal swelling condition as in case of abscess, hernia, hematoma, rupture of urethra or urinary bladder by what is so called exploratory puncture to detect the nature of the resulted fluids if it is :-

A-Blood, so this indicates that this swelling is due to hematoma.

B-Urine, so this indicates that this swelling is due to ruptured bladder or urethra.

C-Pus, so this indicates that this swelling is due to abscess.

D-Ingesta, so this indicates that this swelling is due to hernia.

Treatment:-*1-Treatmente of edema mainly depend upon competing of the main cause.* *2-Resteriction of the salts in the diet*

3-Using of diuretics as Furasemid (strong acting diuretic) or sodium or potassium citrate.

4-Aspiration of the fluid as in case of ascites but it must be done slowly to avoid acute dilatation of the splanich vessels and subsequent peripheral circulatory failure.

3-Hypothermia

Definition:- Hypothermia means that lowering of the body temperature then normal due to either increase the heat loss or decrease in the heat production

Etiology and predisposing factors:-

There are many factors leading to hypothermia

(A)- Factors related to the animals

(B)-Factors related to the environment

(A)- Factors related to the animals:-

1-Decrese of the muscular tone (i.e. decrease in the heat production) as in case of late stage of parturient paresis, acute ruminal impaction, anesthesia or sedation.

2-Profuse diarrhea in cold cow syndrome or vasodilatation shock and reduction of the metabolic activities in the late stage of many diseases are common cause of hypothermia.

3-Decrease in the insulator as in case of decrease in the wool in sheep

4-Hypoxic lambs during dystocia or premature birth

5-Lambs or calves that come from weak, aged or young dams are highly susceptible to be hypothermic (such dame usually give low quality colostrums)

6-Age of the animals:- young animals up to 48 hours of age are highly susceptible to be hypothermic due to :-

-The animal unable to stand to suckle it's dame

-Low heat production due to small size of the animal

7-Selenium deficiency as in young newly born animals there is metabolism of high amount of adipose tissue to produce heat and metabolism of adipose tissue is controlled by 5-deiodenase enzyme which has two atoms of selenium so deficiency of selenium leads to low heat production and hypothermia

(B)-Factors related to the environment:-

1-Bad management which lead to lack of colostrums especially in the first 24h.

2-Parturation a way from shelter or closed place especially in winter season

3-Exposure to excessively cold air which leads to increase the heat loss .

4-Hypothermia may be a sign post-sheering due to decrease in the insulator

6-Some factors that leading to heat loss as:-

-Rainfall -Starvation -Small depth of the wool or thin birth coat

5-Lack of the maternal behavior after parturition as licking of the newly born animals so wet animals are highly susceptible to be hypothermic specially in twine or triple

NB:-*(I)-Most cases of hypothermia occurred in the winter and spring seasons when the adverse climatic conditions are most likely to occur.*

(II)-Fat and liver and skeletal muscles glycogen are the main source of heat production in the first days of the life due to minimal protein metabolism in this period.

Clinical Symptoms:-*1-Shevering especially with wet animals.*

2-Pale mucous membrane.

3-Cold extremities and recumbancy especially in the late stage.

4-Generally the severity of the symptoms varies according to the degree of the heat and glucose loss as showing in the following table:-

Age	Blood Glucose	*Body temperature*			
		30 C	**25C**	**20C**	**15C**
0-5 Hours	Normal or more than normal	Recumbancy	Unable to stand	Enter in the coma	Coma and death
6-12 hours	Low than normal	Unable to stand	Enter in unconsciousness condition	Coma and death	-

Diagnosis:- *-Case history and clinical symptoms*

-Detection of the body temperature and blood glucose level.

Treatment:- **(A)-In mild hypothermia:-**

-Dryness of the animal and put it in a worm placed and providing the colostrums in the newly born animals.

(B)-In sever hypothermia:-

-Injection of glucose 20% 10 ml./kg.Bwt. -Worming of the animal in worming place

-Giving colostrums 20ml./kg.Bwt. Normally or by using of stomach tube.

4-Anemia

Definition:- Anemia can be defined as deficiency in the circulating erythrocytes which may be due hemorrhage or increase the destruction of RBCs. Or due to decrease the production of erythrocytes and /or hemoglobin.

Etiology:-

Anemia can be classified according to the etiological factors into:-

(A)-Hemorrhagic anemia:-

This type of anemia is usually associated with the conditions of hemorrhage either internal or external types.

(B)-Hemolytic anemia:-

Usually associated with the conditions which lead to excessive destruction of the erythrocytes as in case of:-

1-Babesiosis and anaplasmosis. 2-Water intoxication

3-Chronic copper toxicity 4-Hypophosphatemia

5-Snake venom

6-Treatmente of long acting tetracycline (especially in pet animals and horse)

7-Coccidiosis in calves and yearling animals.

(C)-Anemia due to decrease the production of RBCs or Hemoglobin:-

According to the causes of this type of anemia there are:-

(I)-Nutritional anemia (II)-Chronic disease

(I)-Nutritional anemia due to :-

1-Cobalt and copper deficiency in ruminants

2-Iron deficiency especially in young animals

3-Potassium and pyridoxine deficiency

4-Folic acid deficiency 5-Vitamin A deficiency

(II)-Chronic disease as in case of :-

1-Chronic suppurative condition may lead to anemia due to depression of the erythropiosis.

2-Radiation injury 3-Intestinal parasitism

3-Soybean poisoning or phenylbutazon (anti-inflammatory) which may cause depression in the bone marrow.

5-Temporary anemia for several weeks after sudden movement to high altitude especially in cattle (high altitude disease)

6-Anaemia due to destruction of the bone marrow (A plastic anemia).

7-Pernicious anemia due to vitamin B12 deficiency which leads to maturation failure of the RBCs and in this type of anemia the RBCs is larger than normal and contain greater amount of hemoglobin so it is called macrocytic hyperchromic anemia.

Clinical Symptoms:-

1-Pale mucous membrane is the most characteristic sign and it is varies according to the degree of anemia.

2-Other signs of the causative diseases as in hypophosphatemia or parasitism

3-Muscular weakness and depression with anorexia ion some cases.

4-The pulse rate is increased with high amplitude (tachycardia).

5-Labored respiration due to anemic anoxia

6-Icteric mucous membrane especially in the hemolytic type.

Diagnosis:-

(I)-Case history and clinical symptoms

(II)-Clinical pathology:-

1-The clinical signs of anemia usually not appeared until the hemoglobin level fall below 50% of the normal level as well as 20% is compatible with life.

2-Erhtyrocytic count and PCV usually depressed.

3-Increase the number of immature RBCs in case of hemolytic and hemorrhagic anemia.

4-Total serum protein is increased in case of hemolytic anemia and decreased in hemorrhagic anemia.

5-Serum color usually abnormal especially in hemolytic anemia while it is normal in case of hemorrhagic anemia.

6-In case of nutritional anemia there are:-

A-Low mean corpuscular hemoglobin (MCH =Hb% / RBCs counts) while it is increased in case of pernicious anemia.

B-Low hemoglobin level.

C-Normal erythrothytic count with hypochromasia (decrease the concentration of the color of RBCs).

Treatment:-

(A)-Treatment of the main cause of anemia is essential as in case of hemorrhagic, hemolytic types or in case of anemia due to nutritional deficiency.

(B)- Non- specific treatment of anemia include:-

1-Blood transfusion especially in case of acute hemorrhage or in chronic sever anemia.

2-Hematonic preparations are used in less sever anemia.

3-Iron preparation orally, intravenous or intramuscularly.

4-Using of vitamin B 12 as non specific haematenic preparation or as anti pernicious anemia factor.

5-Sudden Death

*Sudden death is a problem with many economic losses especially in the large scale system and knowing the main cause of the cases of sudden death is very important to avoid this problem.

The causes of the sudden death may be related to single animals or related to a group of animals as the following:-

A)-Etiology of sudden death in single animal :-

(I)Spontaneous internal hemorrhage as in case of

-Rupture of the aorta -Cardiac tamponad

-Oseophegeogastric ulcer -Intestinal hemorrhage

(II)-Per-acute endogenous toxemia:-

-Rupture of the stomach, abomasums or colon especially in young animal leads to peritonitis and sudden death due to endogenous per-acute toxemia.

(III)-Per-acute exogenous toxemia:-

As in case of snake bite but it is rarely to cause sudden death without signs but it may lead to rapid death.

(IV)-Iatrogenic death:- As in case of rapid injection of calcium intravenously or penicillin or in case of rapid infusion in pulmonary Odem

(V)-Trauma:- Specially in the skull which may lead to destruction of CNS or internal hemorrhage.

B)-Etiology of sudden death in animal group:-

(I)-Lightening strike or electrocution:-

This accidentally specially in farm system

(II)-Nutritional deficiency or poisoning :-

This occurred when there is animal group exposed to plants that cause bloat, hypomagnesaemia, nitrite poisoning, copper deficiency which causes acute myocardiopathy (falling diseases in cows)

(II)-Diseases caused by infectious agents as :-

These diseases cause septicemia or toxemia as cattle plague, E-coli, colitis-X in horses.

(III)-Access to potent poisons:-

as in case of lead, cyanide or menonsin or organ phosphorus poisoning.

Diagnosis:-

1-Carful examination of the surrounding environment of the animal

2-History of the injection of some toxic drugs or in toxic doses

3-Carful examination of the dead animal for the signs of

-Struggling -Bloat

- Frothy of the mouth -Paler mucus

-Bleeding from the natural orifices or hemorrhage

4-Necropsy finding and analysis of animal samples for confirmatory laboratory diagnosis of the suspected cause.

6-Stress

Definition: - It is systemic state which developed as a result of long term application of stressors which are environmental factors which stimulate the haemosomatic, physiological and behavioral responses in excess of normal. The only acceptable measurement of the presence or absence of the stress is the blood level of the corticosteroids.

Importance of the stress:-

1-Increase the susceptibility of the. Infectious diseases

2-Bad level of the animal welfare.

3-Reduction of the efficacy of the animal production.

4-Leads to development of the psychomatic diseases.

Etiology:-

1-Road transportation especially if it is associated over crowding of the animals or inclement weather, the transportation considered as major factor of stress associated with high incidence of the infectious diseases in all animals.

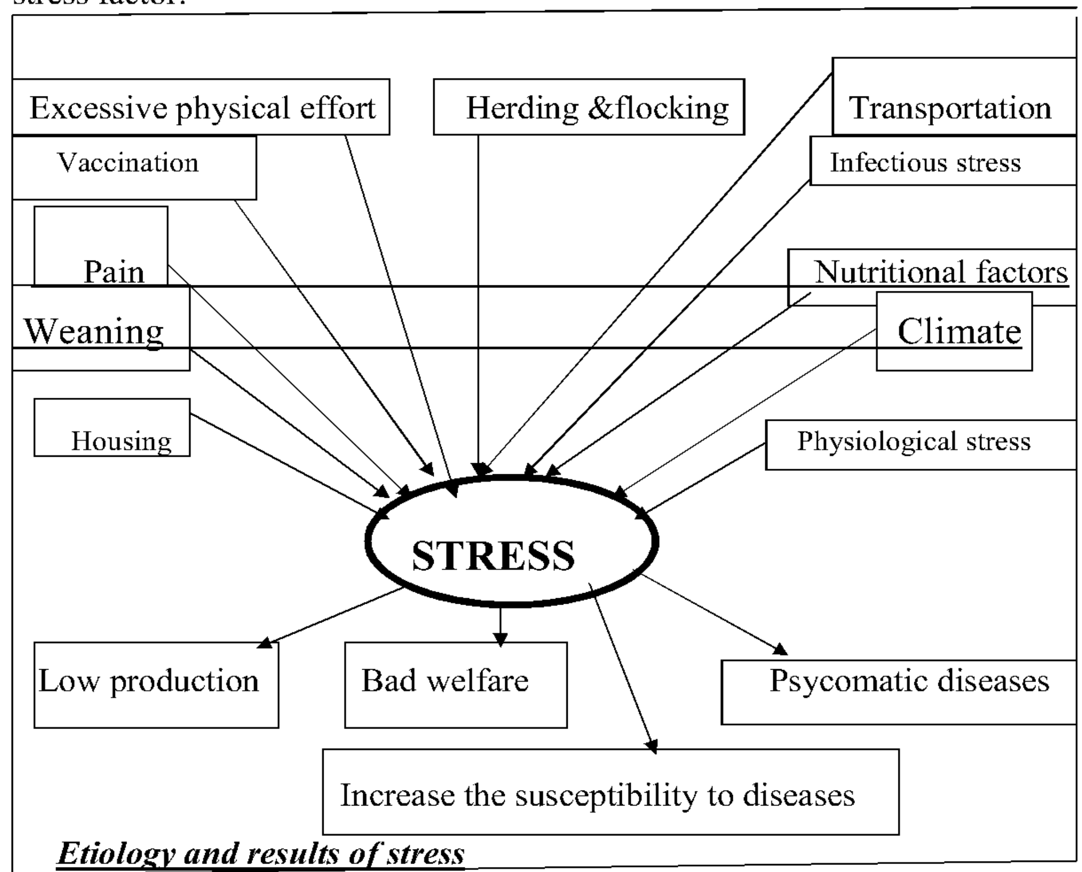
2-Nutritional factors as nutritional deficiency or imbalanced ration (either quantitative or qualitative) or sudden change in the diet considered as stressors that leading to digestive disorders as low level of the immunity.

3-Climate as sudden change in the weather or sudden exposure to the cold or hot weather or living of the animals in a farm with high temperature and humidity and bad ventilated considered as stressors that leading

to what is so called heat stasis or hyperthermia.

4-Excessive physical factors as endurance rides in horses or staggering of the animal during restraining, fear, excitement or handling.

5-Pain as in case of colic, castration, dehorning, docking considered as a stress factor.



Etiology and results of stress

NB. Stressor is any stimulus, internal, external, chemical, physical, emotional that leads to exciting of the neurons of the hypothalamus to release corticotrophin-releasing hormone at rates greater than normal level.

6-Some physiological process as high milk production, parturition and pregnancy so the high producing animal is more susceptible for metabolic diseases more than low producing ones.

7-Vaccination considered as a stress factor due to antigen antibody reaction which usually produce histamine.

8-Herding or flocking as the animals are not accustombred to the new place and the new management system.

Pathogenesis:-

1-Stress is developed when the animal's mechanism which concern with adapting the animal body to the environment are overloaded than normal capacity of it

2-The daily rhythm in the haemostatic or physiological changes as a response of normal changes in the environment require the least form of adaptation.

3-Marked changes as sudden change in weather need a great strain of adaptation.

4-The body system which are principally involved during adaptation to the environment are:-

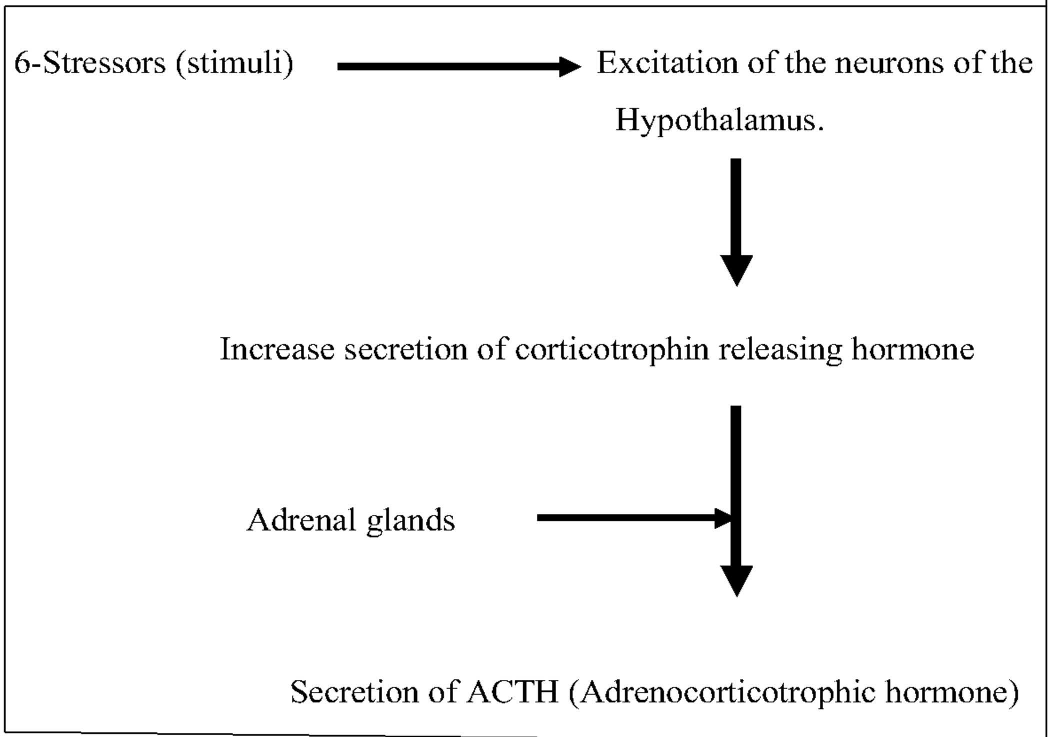
A-Endocrine system for the long term responses

B-CNS for short term responses and for sensory inputs

5-The endocrinal responses against the stress factors are mainly adrenal gland as the following:-

A-Adrenal modularly response related to the flight or fight situation which required immediate responses.

B-Adrenal cortical response becomes operative if the stressful situation persisted for long period.



Management of stress

*Mainly by avoiding all the stress factors.

7-Pain

Definition:-

*Pain is a distressing sensation from stimulation of specific end organ in particular parts of the body.

Etiology:-

*The etiological factors of pain can be classified according to the type or the origin of the pain into:-

(A)-Coetaneous or superficial pain

(B)-Visceral pain

(C)-Somatic or musculoskeletal pain

(A)-Coetaneous or superficial pain:-

In general the coetaneous or superficial pain depend upon the agent which damage the skin as:-

1-Burning or freezing

2-Cutting or crushing by the trauma

3-Sever dermatitis

4-Acute mastitis or laminitis

5-Infected surgical wound

6-Foot rot.

7-Conjuncitivits or foreign body in the conjunctival sac

(B)-Visceral pain:-

1-Inflammation of the serous surfaces, as in case of peritonitis, pericarditis or pleurisy.

2-Distention of internal organ, as in case of impaction, bladder with urine or tempany.

3-Inflammation of internal organs as in case of meningitis, nephritis, enteritis or pelvic cellulites.

4-Swelling of the organ as in case of brain edema.

5-Streching of the mesentery or mediastinal.

6-Colic or spasm in the visceral organs.

(C)-Somatic or musculoskeletal pain:-

1-Tearing and haematoma of the muscles.

2-Myositis and space occupying lesions in the muscles.

3-Osteomylitis, fracture, arthiritis or joint dislocation.

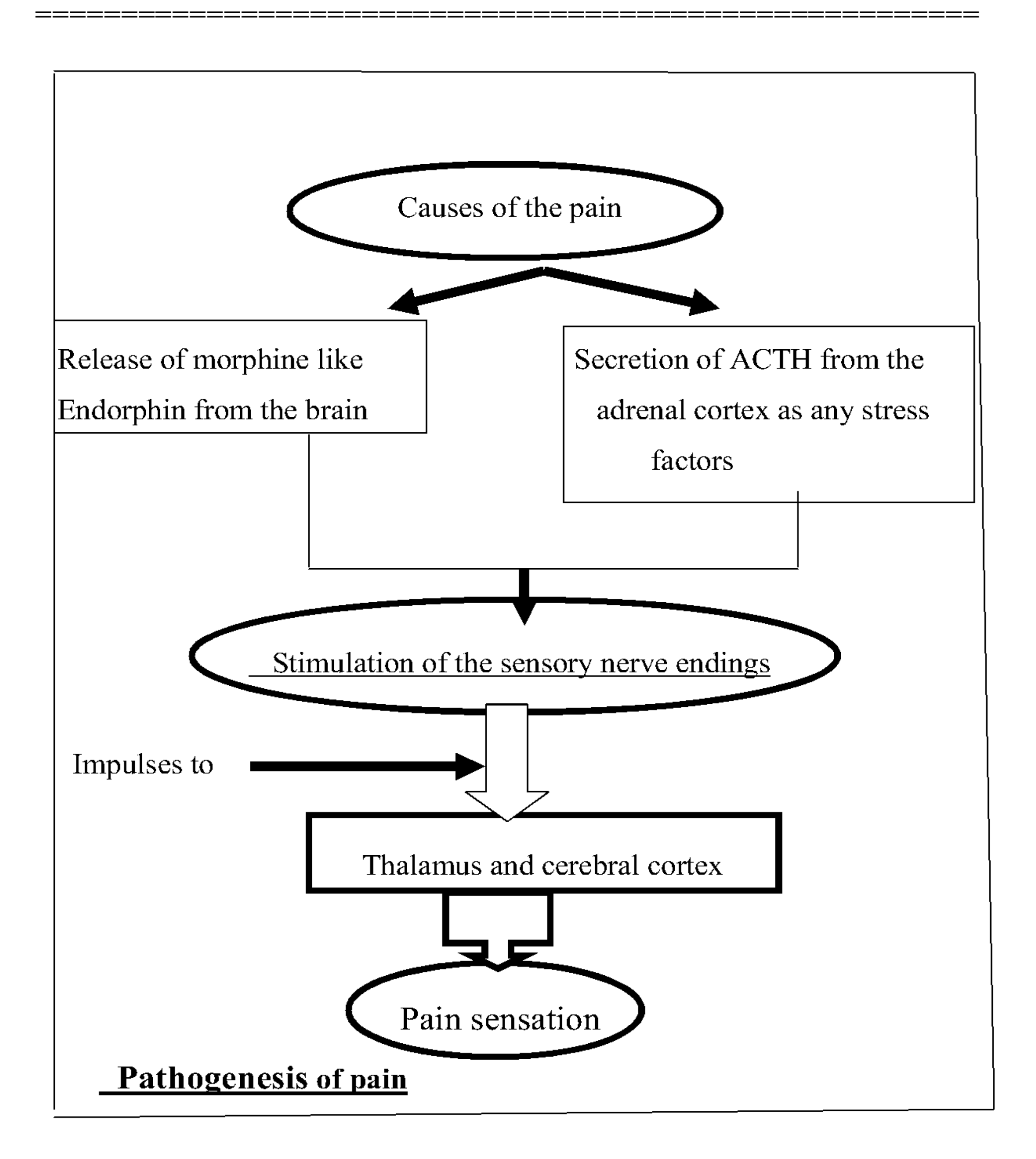
4-Sprain of the ligament and tendons.

5-Ischemia or generalized tetany of the muscles.

6-Tail docking, castration or dehorning or other wounds.

Pathogenesis:-

As the pain receptors are distributed as end organs in all body systems and organs and such receptors are connected to the CNS by the sensory nerve fibers so, as shown in the following diagram:-



Pathogenesis of pain

Clinical Findings:-

(A)-Physiological responses:-

1-Tachycardia, polypnoea and papillary dilatation

2-Hyperthermia and sweating.

3-Dehydration, acid base imbalance and endotoxic shock are present.

(B)-Behavioral responses:-

1-Abnormal posture and gait especially when the pain is somatic (musculoskeletal) as in the form of:-

-Lameness -Rapid shifting of the body weight from one leg to another

-Abduction of forelegs (chest pain) or hind legs (abdominal pain)

2-Rolling, pawing or grinding on the teeth especially when the pain is visceral

3-Animal activities may be related to the site of the pain as flank watching as in case of colic or abdominal pain

4-Gringing on the teeth with head pressing as in case of increase of the intracranial pressure such as in case brain edema or lead poisoning.

5-Vigrous licking of the skin as in case of urticaria or dermatitis.

Diagnosis:-

(1)-Ensure that, pain is the main cause of the observed sings.

(2)-Detection of the pain by the veterinarian by:-

-Pressure, palpation or percussion on the suspected area.

-Movement by having the animal walk activity or by passively flexing or extending the limbs or neck.

-Stimulation of pain as inducing the cough by digital pressure on the larynx or trachea

-Relief of the pain by correction of the lesion (therapeutic diagnosis).

(3)-Specific test as in case of traumatic pericarditis or traumatic reticuloperitonitis by using what is so called ′′***pain test***′′.

(4)-Detection of the blood level of cortisone.

(I)-Relief of pain in human act

(II)-***Treatment:-*** Analgesia should not be used (specially in case of somatic pain) so as it obscure the clinical signs which may be necessary to observe, to properly diagnosis or maintain surveillance of case.

(II)-It may be necessary to protect the animal from massive self-injury

(IV)-Analgesia for visceral pain is readily available and relatively effective as: - -Ethyl alcohol -Phenylbutazon

-Salicylate -Xylazine -Flunixin -Cholarahydrate

A-Ethyl alcohol:- by epidural injection to prevent the straining

B—Salicylate:-as Aspirin or acetylsalicylic acid

-Dose: - 100mg/kg Bwt. Orally or every12 hours

-Salicylate is slowly absorbed from the small intestine, so it can be given intravenously (35 mg./kg. Every 6 hours in cattle or 25 mg/kg. But every 4 hours in horses).

-Salicylate acts by blocking the pain mediators which stimulate the pain end organs but it have not central depressant effect on the thalamus or cerebral cortex.

C-Phenylbutazon:- 1-Used specially in musculoskeletal pain in horses

2-Gives good results specially in mild or moderate pain conditions that caused myositis, laminitis and so on.

3-The half life of the drug in the plasma is about 3-5 hours so that repetition of the treatment is recommended.

4-Oral or intravenous rout give results faster than that the intramuscular rout (dose:- 4.4 mg/kg. Bwt. Daily for 5 days).

NB.:- Prolonged use of phenylbutazon especially in ponies or newly born animals at dose of 10-12 mg/kgBwt. Daily for 8-10 days may lead to ulceration of the alimentary tract.

5-Dose in cattle is 10-12 mg/KgBwt. Followed by maintenance dose of 2.5-5mg/kgBwt.

D-Flunixin meglunine:- Non-narcotic, non-steroidal anti-inflammatory and analgesic drug(1.1mg./kgBwt. Give results within 12 hours for about 30 hr)

E-Chloral Hydrate: - 3-4 gm. /50 Kg.Bwt. 10% solution intravenously.

(V)-Supportive treatment:-

1-Hot fomentation or cold bath on the affected lesion is necessary to relief the pain

2-Providning of good quality and quantity food and water and bedding to avoid self injury.

3-Walking may give good results as in case o colic.

4-Fluid therapy and Dextrose.

8-Organophosphrous Toxicity

(I)-Organophosphate insecticides:-

1-Prohibtion and reduced use of the chlorinated hydrocarbon insecticides has resulting in increase the use of the organophosphates and carbamate insecticides as Diazinon, Malathion, Trichlorfon, Ronnel or Parathion

2-The vast majority of the insecticides toxicities observed in the domestic animals are usually due to use of one of the mentioned insecticides.

3-The insecticides may be applied topically as powder or spray , used as bathing chemicals and administered orally for internal parasites control.

4-The organophosphates or carbamates insecticides exert their influence by reversible or irreversible binding of the acetylcholinestrase enzyme, so it permitting continuous cholinergic stimulation by excessive liberation of the acetylcholine enzyme (i.e. the organophosphates insecticides have parasympathomimetic actions).

5-The effect of the organophosphates insecticides is much more toxic for the insects than they are to the animals.

Clinical Symptoms of organophosphates toxicity:-

1-The clinical symptoms of the organophosphates insecticides may be appeared within 24 hours to two weeks after application of the insecticides.

2-Increase the lacrimation

3-Excessive salivation and diarrhea

4-Ataxia, incoordination and muscular tremors

5-Paralysis and convulsion

6-Respiratoy interferences due to the bronchoconstrictor effects of the organophosphate insecticides.

7-Pulmonary edema and muscular fasciculation

8-Terminal signs (related to the nervous system and respiratory system) are sever and death usually due to the respiratory failure.

9-One or more of the mentioned clinical symptoms may be appeared on the same animal depend on the doses, and the rout and the time of application of the insecticides.

Treatment:- Mainly directed to :-

(I)-Chemically antagonizing the excessive acetylcholin that presented in the nerve synapses.

(II)-Increase the liberation of the cholinesterase enzyme.

(III)-Providing the respiratory assistance to avoid the death of the respiratory failure.

1-All toxicities that caused by this group of the insecticides should be immediate treated by the intravenous administration of the atropine sulphat (0.5 – 0.05 mg of the active principle of atropine sulphat /Kg.Bwt.) or 1ml. (atropine sulphate 1%)/20-200 kg.Bwt. (IM, S/C or IV).

2-Mydriasis (dilatation of the eye pupils) and absence of the oral salivation indicated to the atropinization and response of the treatment.

3-Repetation of the treatment by the atropine sulphat is recommended if necessary.

Animals which not response to the atropinaization usually not suffering from toxicity and it may be suffering from other diseased condition so complete history should be taken completely and thoroughly clinical examination of the animal should be applied

4-Artifical respiration by messaging of the chest is recommended

5-Supportive treatment as high amount of fluid therapy IV is recommended also specially in severally affected animals.

6-Avoiding the application of the insecticides specially in bad weather conditions or other stress factors

7-Avoid application of the un-necessary surgical operation in the season or time of application of the insecticides.

8-Cleaning of the skin by the tape water in case of application of eh insecticides by the spray or powder applications.

9-SELENIUM TOXICITY(Selenosis)

Selenium toxicity is a serious threat to the livestock, there is more data on the toxicity of selenium in sheep, but seleniosis affects all livestock. There are two general types of toxicity, acute and chronic.

A)-Acute toxicity:-

(I)-Acute toxicity is caused by the consumption, usually in a single feeding, of a sufficient quantity of highly seleniferous plants, or administration of overdose during the treatment of selenium deficiency, which produce sever symptoms,

(II)-Usually, death occurs within a few hours.

(III)-Sheep and goat are the most likely species to be affected.

(IV)-Many studies have been shown that possibly as little as 3mg/kg body weight is the minimum lethal dose,

(V)-Signs include:

-Abnormal movement
-Dark watery diarrhea
-Elevated body temperature
-Weak and rapid pulse, Labored respiration,
-Bloating and abdominal pain
-Mucous membranes are pale and blue
-The pupils are dilated.

(VI)-There is no known treatment to reverse the effects of the poisoning, and sometimes the animals dies before a diagnosis can be made.

B)- Chronic toxicity:-

(I)-There are two different types of chronic poisoning dependent on the chemical form of the ingested or administrated selenium. "***Blind staggers***

"occurs when animals ingested or administrated water-soluble selenium compounds naturally found in accumulator plants. Toxicity from eating plants or grains with protein bound, insoluble selenium, is called '"***Alkali disease".***

(II)-***Blind staggers***:-

*Normally occurs in the sheep feeding on seleniferous plants and it is characterized by three stages: -

1-Wandering, stumbling over objects, anorexia and visual impairment.

2-Increase the severity of the first stage, front legs seem unable to support animal

3-Blindness, paralysis of the tongue and swallowing mechanism, rapid and labored respiration, salivation and low in the body temperature.

(III)-The animal will die within a few hours from the onset of the third stage.

(IV)-The action of the toxicity has been documented to delay between stages.

(V)-The first and second stages may be unnoticeable, and then weeks later; the animal may show the signs of the third stage and die. In sheep, it is more difficult to diagnose because the stages are not as well defined as in cattle.

(VI)-Toxic amount of selenium can also cause birth defects in offspring from the dame fed or administered such toxic amount of selenium.

(VII***)-Alkali disease***:-

*Is more chronic than blind staggers, often taking years to manifest itself. It is caused by feeding on plants and grains that have protein-bound, insoluble selenium.

*This disease can affect all livestock. General symptoms include: lack of vitality, anemia, emaciation, and stiffness of joints, lameness, and rough coat, loss of long hair and hoof sloughing and deformities. Hoof deformities are a classic sign of selenium toxicity and can cause lameness and sever pain for the animal.

(VIII)-The most effective way of preventing selenosis is to remove the animals from the seleniferous area or using the recommended doses of the selenium preparations during the treatment of the selenium deficient cases.

(IX)-Treating the soil with sulfates, thus changing the sulfur: selenium ratio, can sometimes depress selenium uptake by accumulator plants, Results from many studies have shown that feeding a higher protein diet may reduce the toxicity of selenium, animals fed the same amount of toxic selenium but fed a higher protein diet lived for a few more days than those animals fed low protein diet.

(X)-Dilution of high selenium feed with low selenium feeds in mixed ration will help to prevent toxicity.

10-Snake Bites

***Symptoms and danger signals*:-**

The risk of snake bites depends on many factors, such as:

1-The species and size of the snake.

2-The amount of venom injected.

3-The number of bites.

4-Individual sensitivity to the venom

5-The localisation of the bites (bites in the head or on the body are most dangerous, but the bites will typically be on arms or legs).

6-The weight of the victim (most dangerous for children).

7-The general state of health of the victim.

Symptoms with rapid onset:

1-local pain, swelling and discolouration at the site of the bite are to be expected, but may not arise immediately after the bite (for example the bite of the coral snake will rarely cause immediate local reactions), and the general reactions often do not appear until 8 to 24 hours have elapsed).

2-Within the first 10 to 15 minutes to a few hours after the bite has occurred, general symptoms may appear such as a sense of anxiety, malaise, vomiting, dizziness, bouts of sweating, respiratory distress, bleeding, heart failure and shock, muscle contractions, confusion, convulsions, paralysis, unconsciousness and death.

<u>Symptoms with later onset (often 6 to 24 hours after a bite):</u>

1-Local (around the site of the bite): increasing and massive swelling, blistering and bleeding often occur in the skin and tissues just below it, and muscles.

2-Blood clots may occur in the surrounding blood vessels.

3-Necrosis (tissue death) of skin, connective tissue and muscles is an evident risk.

4-General symptoms: increasing grogginess, vomiting, respiratory difficulties, fever, falling blood pressure and shock.

5-Bleeding from the mucous membranes (e.g. the gums), bloody vomit and stool and blood in the urine may also occur.

6-Disturbances of sensation or paralysis may occur, often first in the face and later in the muscles involved in swallowing and breathing.

<u>In principle, snake venoms act in three 'different' ways:</u>

1-Haemotoxins, i.e. venoms that split (haemolyse) the red blood cells, or affect the ability of the blood to clot (coagulate).

2-Neurotoxins, i.e. venoms that in particular paralyse nerve transmission to the muscles and in the worst case paralyse the muscles involved in swallowing and breathing.

3-Cardiotoxins, i.e. venoms that have a direct harmful action on the heart and lead to circulatory failure and shock.

But as a number of other factors, including possible allergic reactions, are also involved in poisoning, the situation is often far more complex and

unclear, and there are often typically 'mixed' reactions and symptoms.

First aid for snake bites

1-Do not panic. Only a few poisonous snakes are really dangerous to humans and animals (about 15 %). In addition, often no venom will be injected with the bite. It is estimated, for example, that venom is injected in only 20 to 30 per cent of rattlesnake bites and in 50 per cent of bites from coral snakes.

2-Avoid all unnecessary movement to prevent any venom from spreading in the body.

3-If possible, wash the site of the bite quickly and carefully with clean (or boiled) water and soap.

4-Spittle from Asian and African spitting cobras must be washed away immediately from the eyes and mucous membranes to prevent it from being absorbed into the body.

5-A firm bandage (wide tourniquet) can be placed over and, if necessary, around the site of the bite which may reduce the uptake and spread of the venom, but it is important that a pulse can continue to be felt on the 'far' side of the bandage.

6-It is important (as always) to keep the airways free of mucus, vomit and blood.

7-Ensure that the victim or affected animal is transported as quickly as possible to a doctor or hospital.

8-In human, the patient must be driven or carried, and if the person is suffering from nausea and vomiting they should be made to sit up or be

placed in the 'recovery' position, i.e. lying on their side, to prevent any vomit from going down into the airways and lungs. Many snake venoms act quite slowly (4 to 20 hours after the bite), but this should not delay transport, as other factors may also be involved (e.g. allergic people or children in whom the venom may act more rapidly).

8-There is often an urgent need for treatment with oxygen, attachment to a drip and possibly anti-shock treatment.

9-Antiserum treatment (expensive) may be appropriate and life-saving, but should always be overseen by a doctor.

11- Copper Toxicity

1-Copper play an important role in melanin, ,elastin and collagens production as well as in immune system and participate in the structure of many metal enzymes as cereloplasmine as mentioned before in copper deficiency.

2-Noraml copper level in the diet is 8-11 ppm. And the toxic dose in the diet is 15-20 or more than 25 ppm. that may leads to copper toxicity.

Causes:-

1-Administeration of high doses of copper during treatment of case of copper deficiency

2-Feeding on diet contain high concentrations of copper for long period.

3-High copper: molybdenum ration more than 10:1

4- Zinc deficiency or lack of vitamin C or manganese.

5-High copper level in the soil or in the drinking water.

Clinical signs:-

1-Generally both sexes are susceptible for copper toxicity and it is more common in the young aged animals.

2-Weakness with yellow mucous membrane due to haemolytic anaemia caused by copper toxicity.

3-Heamoglobinuriea and the urine may take dark brown colour.

4-Diarrhoea and increases heart rate.

5- Pm. Lesion the liver appeared pale and dark greenish black kidney.

Diagnosis:- mainly through clinical signs and history of the cases and confirmed by detection the copper level in the blood or RBCs copper or determination of cereloplasmine.

Treatment:-

1-Copper should be removed from the diet or change the animal pasture.

2-Addition of molybdenum or sulphate as it reacts with copper and forming non absorbable complex and decrease the rate of absorption of copper.

Addition of vitamin C or zinc also decreases the absorption of copper.

3-Electrolytes and fluid therapy for flushing the kidney.

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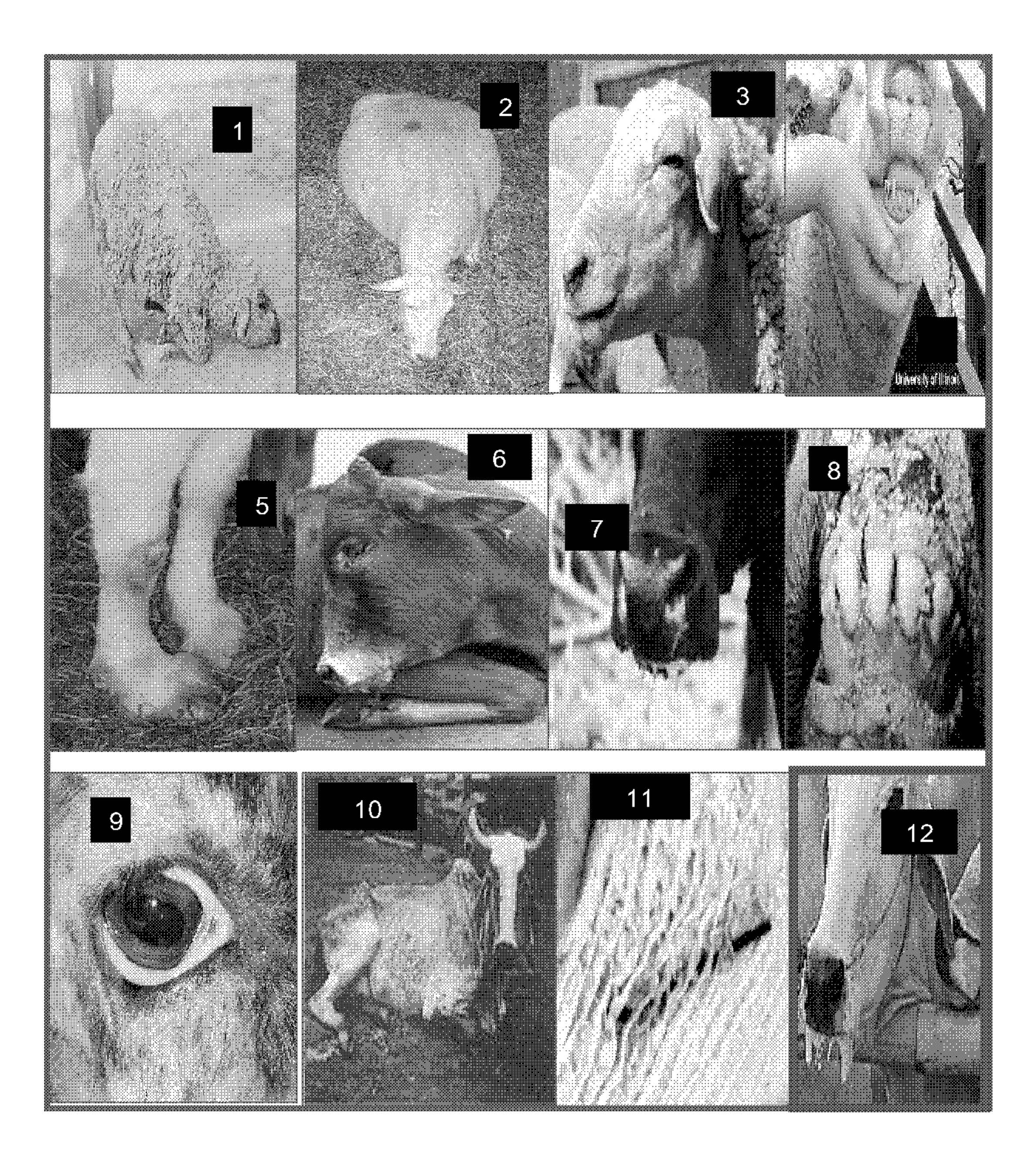
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